This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

TRANSACTIONS OF THE

SECTION ON PRACTICE OF MEDICINE

of the
American Medical Association
at the Fifty-fourth Annual
Session, held at New Orleans,
May 5 to 8, 1903 :: :: :: :: ::

AMERICAN MEDICAL ASSOCIATION PRESS
CHICAGO: NINETEEN HUNDRED AND THREE

BOSTON MEDICAL
MAR 16 1928
LIBRARY

CATALOGUED
MAR 16 1928
7045

OFFICERS FOR 1902-1903

CHAIRMAN.

W. S. THAYER - - - - - BALTIMORE.

SECRETARY.

JAMES B. MCELROY - - - - STOVAL, MISS.

EXECUTIVE COMMITTEE.

GEORGE DOCK, ANN ARBOR, MICH.
J. M. ANDERS, PHILADELPHIA.
FRANK A. JONES, MEMPHIS, TENN.

OFFICERS FOR 1903-1904

CHAIRMAN.

ALEXANDER LAMBERT - - - - NEW YORK.

SECRETARY.

J. L. MILLER - - - - - - CHICAGO.

EXECUTIVE COMMITTEE.

J. M. ANDERS, PHILADELPHIA.
FRANK A. JONES, MEMPHIS, TENN.
W. S. THAYER, BALTIMORE.

CONTENTS

PAGE.

Observations on the Teaching of Clinical Medicine. W. S. Thayer 9

The Boy's Venereal Peril. Ferd. C. Valentine..... 20

Continued Fever—Neither Malarial Nor Typhoid. T. J. Happel 31

Southern Fevers. William Krauss 42

A Clinical Observation of Ninety Cases of Typhoid, with Four Deaths, with Special Reference to Therapeutic Fasting. R. M. Harbin 52

Typhoid Fever in Colorado. A Report of Five Hundred and Twenty-nine Cases. J. N. Hall and C. E. Cooper.... 61

Weakness and Dilatation of the Heart in Chronic Nutritional Disease. G. W. McCaskey 74

Limitations of Non-Surgical Treatment in Acute Intestinal Obstruction. J. Rilus Eastman 87

Occurrence of the Strongyloides Intestinalis in the United States. M. L. Price 100

Clinical Diagnosis of Intestinal Parasites. C. W. Stiles.. 137

The Treatment of Uncinariasis. T. A. Claytor 142

Bacillary Dysentery (Shiga). C. F. Mason 161

A Contribution to the Study of Summer Diarrheas of Infancy. J. H. M. Knox 176

Malarial Dysentery. W. B. Burns 197

Pathology and Clinical History of Amebic Dysentery. H. F. Harris..... 201

A Study of the Cases of Amebic Dysentery Occurring at the Johns Hopkins Hospital. T. B. Futcher..... 213

The Treatment of Acute Dysentery. J. M. Anders..... 244

Abdominal Pain in Pleurisy and Pneumonia. J. B. Herrick 259

Traumatic Pneumonia. W. T. English 277

The Transmission of Yellow Fever. James Carroll 291

The Stegomyia and Fomites in Yellow Fever. S. F. Chaillé 308

PAGE.

Arthritis Deformans. The Report of a Series of 110 Cases from the Johns Hopkins Hospital. T. McCrae 331

Albuminuria in Diabetes Mellitus. Its Importance as a Factor in the Causation of Diabetic Coma. A. R. Elliott 380

The Passing of Chronic Rheumatism. J. J. Walsh..... 393

Hemoglobinuric Fever. Its Causes and Treatment, with Especial Reference to the Use of Quinia. W. Shropshire 407

Some Clinical Aspects of Tubercular Peritonitis. J. Eichberg 429

Tuberculosis in the Negro. Seale Harris 447

Should the Tuberculous Patient Know the Truth Regarding His Condition? C. P. Ambler 464

Typhoid Fever. Some Unconsidered Hindrances in Its Prophylaxis. J. S. Fulton 470

Sources of Plague. W. J. Calvert 506

The Prophylaxis of Plague. J. J. Kinyoun 516

Executive Session 549

Index 555

OBSERVATIONS ON THE TEACHING OF CLINICAL MEDICINE.

CHAIRMAN'S ADDRESS BEFORE THE SECTION ON PRACTICE OF MEDICINE AT THE FIFTY-FOURTH ANNUAL SESSION OF THE AMERICAN MEDICAL ASSOCIATION AT NEW ORLEANS, MAY 5-8, 1903.

BOSTON MEDICAL LIBRARY MAR 16 1928

WILLIAM SYDNEY THAYER, M.D.
BALTIMORE.

A hundred years ago the young American republic bought from the French government that great tract of land of which the little frontier city of Nouvelle Orleans was the chief settlement. It is a happy chance that brings us together on the centennial anniversary of the Louisiana Purchase in this metropolis of the south.

One hundred years—a small space of time in the world's history, and yet what events have been crowded into this little period! A hundred years ago the country west of the Mississippi was a wilderness. To-day we meet, a national body, the majority, perhaps, of our members coming from what was then forest and prairie, inhabited only by savage and wild beast. And the young democracy, then all but unknown to the powerful empires of the east, has become the dominating factor in the markets of the civilized world.

In this amazing growth and expansion, in this tremendous accumulation of wealth and strength and power, it was scarcely to be expected that our contributions to science would keep pace with those of the Old

World, where accumulated wealth, settled conditions and well equipped institutions of learning naturally tempt the studious and contemplative mind to problems of science and research. And yet, how many of the great discoveries and inventions of the century have come from this new country! In our own branch of learning the record is one of which we may well be proud. The discovery of anæsthesia, the demonstration by Holmes of the contagiousness of puerperal infection, the studies by the students of Louis on typhoid fever, the accomplishments of Bigelow and Sims and McDowell and, lastly, the noble work of Reed, Lazear, Carroll and Agramonte in Cuba and of those who have demonstrated to the world the practical value of their great discoveries—these are but a few examples of an honorable record.

And during this period what a change has come over the condition of medical education in this country! One hundred years ago there were three schools of medicine in America: the Department of Medicine of the College of Philadelphia, the first school of medicine in the United States; the Medical School of Harvard College, and the Dartmouth Medical School of Hanover, N. H. To-day there are over one hundred and fifty.

One hundred years ago only the most favored and fortunate were able to receive anything like a systematic medical training. Not that there were not educated physicians in America. There were in Carolina and New England, in New York and Virginia and Pennsylvania men who had profited by the inspiration and opportunities offered in the best schools of Europe. Many a young man, however, was obliged to begin his studies by "reading medicine" in the office of some older practitioner. His theoretical knowledge was gained from a few books, his main instruction came through the practical observation of disease, gained by assisting at the operations and visits of his preceptor.

In some of our early medical schools the requirements were, however, such as may well surprise those of us

who consider the fact that within fifteen years medical schools in this country were giving a degree of doctor of medicine for one year's study. "Although," says Thatcher in 1828 (American Med. Biography, 80, Boston, 1828) "there is no uniform standard of attainment established in order for graduation, in most of our schools it is required that before a student can be admitted to an examination for a degree he must have attained to the age of 21, have studied three years with some regular physician, attended two full courses of lectures on the different branches of medicine, and, if he has not enjoyed the advantages of a college education, he must furnish satisfactory evidence of having made respectable classical attainments, and particularly that he has acquired a competent knowledge of the Latin and Greek languages, has studied mathematics, natural and experimental philosophy, geography and belles lettres. In several of our schools it is required that he shall have attended the clinical practice of some infirmary for a specified term. It is also required that before he can receive his degree he must pass a close examination in the different branches of medicine and write and defend a thesis on some medical subject."

The first schools of medicine were, for the most part, founded in connection with hospitals, and the actual study and observation of the patient was an important part of the student's curriculum. The multiplication of medical schools, however, proceeded with great rapidity. The number of students in some of these institutions was so great as to preclude the possibility that each individual should have sufficient practical experience from the amount of clinical material at hand, while in other instances schools giving the degree of doctor of medicine were established entirely independently of hospitals or infirmaries. Ten years ago the requirements for admission to many of these schools were almost *nil*. It was not uncommon to hear that this or that good fellow whom one might have known but a day before

as the driver of a horse car, had taken his degree in medicine. On investigation it might be found that for the past one or two years he had been attending evening classes, and at the end of this period had stepped from the platform of his car to the bedside of the patient with a degree attesting that he was a competent practitioner of medicine.

Within thirteen years I remember to have heard a colleague relate with pride that he had just accomplished in one year his whole course in medicine at a university with an old and honorable name, practically without clinical advantages. This is, to say no more, far from the ideals of our ancestors.

In the last few years, however, great changes have come into our methods of teaching. Four years' actual work in a medical institution are everywhere required. In our better schools anatomy, physiology, pathological anatomy, pharmacology are no longer taught as they used to be, by lectures to an untrained body of men. In many instances the student begins his medical studies with a training which has already taught him to think and investigate for himself; and our duty as teachers is coming to be rather that of guides and leaders than of simple lecturers.

The teacher to-day must be a student and investigator in his own line. The time is passing when the professor of anatomy or physiology can be a practitioner of medicine. His whole time must now be given to his studies and his teaching, and his immediate duties are not so much to lay facts before his students as to show the student how to find them out for himself.

And so it is in all these branches. The student in the laboratory sees, acts, controls, verifies. While of old he was expected and required to believe what was said to him, he is to-day taught rather to doubt that for which he can not obtain proof. And in like manner throughout the greater part of the course the didactic lecture is passing—such matters the student can read—

and is being replaced by practical anatomical, physiological, pathological, pharmacological, experiment and experience. The same methods have come into the teaching of physical diagnosis in its restricted sense, as well as of minor surgery—lectures and demonstrations have largely given way to practical experience in small sections. In some of our schools the teaching in these branches compares with the best that can be found in the world.

But when we arrive at the last stage, the study of pathological physiology in the individual, the observation and care and treatment of the patient, we find, strangely enough, that the advances which have been made in our methods of instruction have not kept pace with those in the other branches. If there be one opportunity in the course of the study of medicine which it would seem most necessary for us to provide for our students, it should be the privilege of watching and studying at the bedside those diseases which they are most likely to meet with in practice. It was the first necessity recognized by our ancestors; it is the first necessity that would be appreciated by any non-medical man; and yet I believe that any one who investigates the matter will realize that it is one of the weakest points to-day in our American system of medical education.

What actual clinical experience does the average medical school of this country offer?

The clinical instruction offered in most of the better schools in this country consists in:

1. Exercises in physical diagnosis proper, which may include good practical work in small sections, but which commonly consist more of lectures and demonstrations than practice and experience.

2. Clinical lectures in which but few, if any, of the class approach the patient.

3. Ward visits which are too often infrequent.

4. Exercises in which students report individually on cases which have been assigned to them for study.

The requirement of a continuous service of students

as assistants in hospital wards is almost unknown in this country.

The student who has graduated from such a school may, and often does, leave the institution with a fairly good training in methods of purely physical diagnosis. But how few of us in this room at the time we left the medical school, had ever followed a case of typhoid fever, pneumonia, measles or scarlet fever from beginning to end. And yet there is but one way by which one can learn to recognize and treat these diseases—and that is by bedside experience. Clinics, occasional visits, books and lectures, these weigh but little in the balance. If, as would seem to be the case, the medical records of our late war with Spain reveal a strange lack of familiarity on the part of many physicians with such important diseases as malarial and typhoid fevers, this is due largely to the failure of our medical schools to offer them the necessary bedside experience in the course of their early training.

And again, how few of us as students ever had brought practically before us the vital questions of when and how to administer stimulants in acute infectious diseases; whether, in any given emergency, those drugs should be given which raise or depress the blood pressure. But these are matters which may mean the life or death of the patient, and it is a sad fact that nothing is more common than the administration of nitroglycerin in conditions of profound vasomotor collapse by conscientious men who have missed in their education just that which early and well-directed clinical observation and experience might have brought them. This defect in our medical teaching is further emphasized when we compare the curriculum in most of our schools with that which is required in England and France. In London and Edinburgh, for instance, schools require of a student at least six months' service as a clinical clerk in the medical wards and a similar length of time as a dresser in the surgical department of the

hospital. In Paris the student is required to give his whole morning to ward work of this nature for two years—one year in medicine, one in surgery.

That it is desirable that we in America should offer in our schools and require for our license to practice an amount of practical experience equal to that required in Europe is obvious.

Of the necessity for such requirements if we wish to keep pace with the progress of the world and properly educate our medical men there can be no question.

Of their practicability there can also be no doubt when we consider that such methods have been in force for many years in a country where individual liberties are as jealously guarded as they are in England.

Why is it, then, that so serious a defect exists?

The reasons are various and not altogether simple. The more important would seem to be:

1. The absence of state or national laws requiring such work.

2. The multiplicity of medical schools and the existence of many such institutions unconnected with hospitals of sufficient size to furnish material for the instruction of the students.

3. A wide-spread prejudice existing in many hospitals against the admission of students to the wards and against the use of patients in general for purposes of instruction.

1. The first of these reasons is, of course, the most important. Until the states require such experience for a license to practice there will always be schools which will give a degree for less work. It is for us individually as physicians and collectively as we are associated in teaching bodies to strive to raise the standard and to hasten the day when such requirements are put in force.

2. It is the absence of these requirements which is, in great part, responsible for the multiplicity and inferior

quality of some of our medical schools. With proper state regulations institutions unable to offer the requisite amount of bedside experience would of necessity go out of existence. The hospital is the most important part of a school of medicine, and that school which does not own or, to some extent, control a hospital sufficiently large to furnish practical experience for its students is not only incomplete, it is a pernicious and dangerous institution. It is greatly to be hoped that state or national laws, as well as the force of enlightened public opinion, may soon remedy the evils caused by the existence of such establishments.

3. As a matter of fact, however, most even of our larger and better supplied institutions do not, as they should, own or wholly control the hospitals on which they depend for clinical material, and the vague fear that the admission of students to the wards may prove prejudicial to the interests of patients or institution deprives these schools of an enormous amount of valuable material. That this fear is wholly without foundation is a fact which is clear enough to any one who has had experience with active ward teaching. But there is another side of the question, to which attention has recently been called by Dr. Osler,* which is of very great importance to the hospitals themselves. The duties of the house physician with the recent advances in methods of precision in diagnosis and study of disease in ward and laboratory, have in latter years increased to a degree that demands outside assistance if the patient is to be properly studied, properly watched, properly treated. Much of this work can be done better by advanced students than by any other class of assistants. To assign three or four advanced students as clinical clerks to a ward of twenty-five or thirty patients, letting each student be responsible for the history, the examination of the secretions, the general observation of four or five patients, under the control of the ward physician, is to

* Med. News, N. Y., 1903, lxxxii, 49.

give the physician invaluable assistance in the keeping of records, in accuracy of observation, in the detection of many points in the progress of the disease, points which may be, and often are, of vital importance to the patient. The admission of students to the wards of hospitals in this capacity is not only without harm to the patient but is of the greatest practical value. That the careful study of each case resulting from such a system is, moreover, without annoyance to the patient I can testify from my experience of the past thirteen years in Professor Osler's wards, in which I have seen the direct transition from one system to another. To the student this experience is invaluable, and it seems to me to be absolutely necessary, if the degree of doctor of medicine is supposed to represent a qualification for practice.

Such experience, however, is of limited value unless it be associated with frequent and careful ward visits from the instructor. And these visits make considerable demands on his time. Teaching, direction of the research work of the department, the study of the material afforded by the clinic, these should be the main duties of the professor of medicine—duties incompatible with other than a purely consulting practice. To make this possible our universities must recognize the fact that the professorship of medicine, as well as the professorship of anatomy, should be properly endowed. Only by the adoption of some such system as this can we hope to bring our instruction in the practice of medicine to the level of that which is given in Europe.

For proper ward teaching the clinic must be so organized that the professor shall have several regularly appointed assistants, who shall take part in the investigation and instruction going on in the department. This is desirable, not only for the instruction of the students, but for the education of teachers. If we are to approach the infinite number of problems in pathological physiology which come before us every day in the observation

of disease in anything like the manner in which such problems in normal physiology are studied in the laboratory, we must have a regular staff of assistants. No laboratory of physiology, for instance, could accomplish scientific work if the entire staff consisted of the professor and a number of young internes appointed annually from the graduating class. It is only through the opportunities offered by such positions that younger men are enabled to acquire that experience which fits them for the task of teaching.

The problems involved in the study and teaching of clinical medicine differ in no essential from those connected with the study and teaching of physiology, and until we recognize this fact and meet it squarely our clinics will remain unproductive and our teaching inadequate.

The most serious impediment in the way of advance in our methods of clinical study and teaching is the widespread institution of rotating services in our hospitals. In no service where the head of the department changes every three or four months can really valuable investigation be accomplished. And the best instruction can not be given to students in wards presided over only by a visiting physician and young, annually appointed, assistants. A permanent service, even though it be small, and a fixed staff of assistants offer incalculably greater advantages for study and research and teaching.

It is, of course, evident that instruction of this sort in larger schools can not be given in one clinic or by one man. The day must come when every well equipped hospital shall be at the same time a laboratory for the study and teaching of clinical medicine, offering the clinical advantages required by the central university or by state regulation. Every physician or surgeon holding an appointment at a large hospital or out-patient department should be encouraged to teach.

The hospital will gain by the supply of the best trained assistants and by the education of the staff, for

there is no training so good as that of teaching, and there are no such spurs to work and progress as the questions and suggestions of the enthusiastic student.

The student will gain, in a few months, experience which it might require years to accumulate after graduation, and he will acquire that experience, as I have said elsewhere, at a time when he is free from the prejudices and preconceived ideas which are sure to obscure the judgment of the man who has gained his medical experience second hand from books or lectures.

The general public will gain the simple protection which any civilized government owes its citizens.

In this very city there are opportunities such as exist in scarcely any other community of this country for the realization of our ideals in medical instruction—a city of over 300,000 inhabitants, a fine old hospital of nearly 900 beds, a single school connected with a university of honorable past and most promising future. Tulane University has been a leader in this branch of medical education, but it can do more. The university can still offer to the hospital and the hospital to the university that which might make New Orleans the model for the rest of the country.

THE BOY'S VENEREAL PERIL.

FERD. C. VALENTINE, M.D.
NEW YORK.

I. INTRODUCTORY.

Many important books have been written and many valuable papers contributed to medical journals on the dangers of venereal diseases. All show that venereal diseases are more perilous to the individual, the family and the State than are all other ailments combined.

Study and effort directed to their abatement lead to the inevitable conclusion that venereal diseases are due essentially to prostitution, clandestine and public. The knowledge that venereal diseases are due mainly to prostitution does not, however, reveal the means of curing the evil: 1, Because all efforts to reduce public prostitution have proven, and are always likely to prove, futile; 2, because clandestine prostitution is necessarily beyond all efforts to limit its still greater power for evil.

The principal cause for the physical and moral wrecks produced by prostitution lies in ignorance. The same ignorance misleads many of the victims of prostitution to regard lightly the ailments thereby acquired.

The need for public enlightenment being manifest, many earnest men, I among them, have advocated lectures to the people on these subjects. More mature thought has, however, brought the conclusion that such lectures will often fall short of their purpose, principally because, to be effective, instruction must be given at the opportune moment. A state of receptivity dependent on a condition so individual as mental, physical and sex-

ual puberty, in any assemblage, necessarily forms as varying a factor as there are people present.

Obviously the effective teaching of these matters can be done only in private. It would seem at first glance that this is the father's duty. But some, indeed many men are so far removed psychologically from those nearest them that they could never successfully undertake this delicate task. Such men must relegate the duty to the physician, the teacher or the clergyman.

Among teachers and clergymen many have vague, and often gravely erroneous, ideas on these subjects. Indeed, a majority of them has not received even rudimentary education on the structure and functions of the genito-urinary organs. They consequently have no knowledge whatever of the diseases due mainly to misuse of the genesic function.

If, for the reasons given, the father, clergyman or teacher is disqualified from imparting the necessary warnings, it may be well to direct the youth to study the third, fourth and fifth divisions of this paper.

II. THE DANGERS OF VENEREAL DISEASES.

In occasional contributions to our medical journals one eminent man or another asserts that the exercise of the sexual act is unnecessary for the preservation of health. As far as I know this has not been refuted. One reason for this silence may be, and probably is, that such refutation would be more likely to be read than were the articles contested. They would, as all such things do, fall into the hands for which they are least intended and be perverted to serve as scientific endorsement for libertinage and profligacy.

Wiser and deeper students may, in the course of time, establish whether illicit relations between the sexes is ever a vital necessity. For the present it suffices to say that young men are everywhere beset by temptations and induced by the spirit of evil, by the jests of their companions and by the solicitations of mainly clandestine

offenders to seek sexual gratification, the desire for which is thus awakened.

The present purpose would not be advanced by a discussion of the need of sexual gratification for the maintenance of health. Moreover, the subject seems too individual for generalization, and, therefore, not capable of wide application.

So much, however, experience shows, that no matter what volumes may be written on the wisdom of male sexual purity, whatever the endeavors of moralists, there will be, as there always has been, a vast majority who disregard precepts which oppose their desires. In consequence there is, as there always has been and will be, an overpowering multitude of men who acquire venereal diseases to the detriment of themselves, their families and the State.

To what extent this detriment goes is incalculable, as statistics on the subject can not be reliable. It will suffice here to note that conservative authors estimate that one-eighth of all the patients in the hospitals of New York are inmates thereof because of venereal diseases or their consequences.[1]

Those in hospitals represent but a small fraction of the people attainted. The latest study of the question[2] is by a body of physicians appointed by the New York County Medical Society for the purpose. This committee makes the appalling declaration that 200,000 people infected with venereal diseases are walking the streets of our city.

The working and consequently earning power of these people is materially reduced. If every infected person loses only $1 per day, then the loss of earnings from this

1. Specific mention of the works of such eminent writers as Morrow, Sturgis, Tarnowsky, Scott and others, is intentionally omitted here because they are well known to the medical profession. Laymen who may desire further information on these subjects can easily obtain from their physicians the titles of the best works that treat of them.

2. Morrow: Report of the Committee of Seven on the Prophylaxis of Venereal Diseases in New York City. Medical News, No. 25, 1901.

cause alone amounts to *sixty million dollars* per year. This loss is directly or indirectly borne by all, even those who are so inexpressibly selfish as to have no feeling for the sufferings of others.

The heaviest part of the burden rests on physicians, who must give their services gratuitously to over 66 per cent. of their patients. But they do not complain of their unrecognized philanthropy, nor do they advertise it. They exercise it as the most important part of their priesthood.

Men who devote their lives to the relief of misfortunes which bring the deepest woes, are daily astounded at the consummate ignorance that prevails among all classes regarding what is, from the nature of things, a generally tabooed subject. It is not only the uncultured, uneducated man who is the victim of unbridled passions; his cultured, educated and intellectual brother is equally and just as frequently unfortunate. There is no excessive charity in here using the word "unfortunate," for he is truly unfortunate and it avails nothing to consider him otherwise; nor is there any excessive optimism in the conviction that if all men could be informed of the dangers of venereal diseases, fewer would contract them, or having contracted them would neglect them as they now do.

Honest endeavors are continually made, especially by physicians, to enlighten the public on this subject. But their excellent counsel must ordinarily be limited to persons who have already acquired the disease, and for whom the topic has at the time absorbing interest.

The ignorance that prevails on these subjects is far-reaching in its effects. The patient, uninformed of the dangers to his health and life, generally does not seek early and radical treatment for the infection. More frequently still, associating venereal diseases with the only mention of them that has come to his notice, namely, in the advertisements of quacks, he consults one of these harpies or patronizes their congeners, the patent-medi-

cine dealers. Later on, and often too late, he learns that every community has conscientious practitioners who scientifically study these diseases. Only then does he discover that he has wasted time, injured his health and imperiled his life through his ignorance and the shamelessness of those who have taken advantage thereof.

As little as the patient knows of the dangers to himself of venereal diseases, so little is he aware of the menace to his family. He should know at least two facts, which in efforts laid before the profession were elsewhere discussed. These facts are:

1. That of all women who die of diseases of the reproductive organs, 80 per cent. are victims of gonorrhea, of which their husbands imagined themselves cured.

2. That of all children born with seeing eyes who became hopelessly blind within a few days after birth, 80 per cent. lost their sight because of infection by gonorrhea, of which the fathers imagined themselves cured. (This paragraph refers to the time before the Credé method of protecting children's eyes at birth was employed.)

The patient is, as a rule, as ignorant of these frequent results as he is of the danger which venereal diseases are to the community. The need, therefore, of instructing the people at large on these subjects is manifest. As said before, however, the knowledge usually is imparted after the damage is done.

Many physicians, I among them, have striven to aid in combating this most dangerous ignorance. Although the papers written were received in a most cordial and kindly fashion, they could not meet the fundamental defect which prevails, that of preliminary education in those whom physicians must instruct. This shortcoming was made painfully apparent by letters from practitioners in all parts of the country.

Manifestly, then, there is need for explanation to the young which will appeal to them at the most susceptible

age, that of puberty. To meet these evident requirements I have, at various times, attempted to construct an essay that would serve the ends in view. But at almost every line I was confronted by the dread that, by the very writings, the youthful mind might be directed to temptations that might not otherwise beset it.

It was my fortune, at the second conference for the Prophylaxis of Venereal Diseases, held at Brussels early in September, 1902, to receive a copy of Professor Fournier's article addressed "To Our Sons When They Have Reached Their Eighteenth Year." This admirable paper, written temperately, with much wisdom and great tact, indubitably meets the requirements of the sons of intellectual people. The temptation at once presented itself to translate Professor Fournier's paper, but throughout it is permeated with the spirit which makes it so eminently acceptable to the French line of thought. It would not apply as well in our own country, where the Anglo-Saxon manner of thinking and living predominates. Appreciating the need of a similar effort, it is now attempted in the hope that better pens will take up the matter for the benefit of the rising generation.

The proposition, as suggested before, involves the problem, at what period of the young man's life this information should be imparted to him. Those closest to him must be the judges of the opportune moment.

The remainder of this paper consists of:

1. The evidences of awakening genesic tendency.

2. Masturbation and the impulses or defects that lead thereto.

3. The evil influences that mislead youths into immorality.

4. Prostitution, public and clandestine, and the mental and physical destruction they bring.

5. Gonorrhea, chancroid, chancre, and their sequelæ.

6. Advertising quacks, nostrum venders and an ex-

posé of the methods they employ to swindle those afflicted with real or imaginary genitourinary and other ailments.

7. The reasons why a youth with a real or fancied illness due to immorality should consult a physician whom he knows, or whom his father knows.

8. The reason why reputable practitioners do not advertise.

These matters, being addressed to boys, are necessarily presented in such elementary outline and puerile language that they have no place in a scientific publication. The author therefore requests their omission here, but for completeness will add them to the reprints of this article, which will be furnished to physicians on request to that effect.

31 West Sixty-first Street.

DISCUSSION.

Dr. James J. Walsh, New York—The great universities of Europe, especially those in Berlin, have deliberated on this question, and the students are taught what is meant by venereal diseases and what their significance is. This is certainly not a pleasing question for deliberation, but it is an exceedingly important one. At the University of Berlin a course on the significance of venereal disease, open to all the students, is supported out of the emperor's purse, and is very largely attended, thousands of young men attending such lectures. The time must come, and shortly, too, in America when the universities do something to protect the students and give them this much-needed knowledge. At the present time all the universities are necessarily situated in the large cities, and the young men from the country are invited to come to the city, where they meet with the dangers of city life. I think it is our duty to give these young men such knowledge as will protect them against the physical evils of venereal diseases. Faculties which do not make some provision for this are neglecting a serious duty. Any objections to such a course being instituted in our universities are founded on pusillanimous prudery.

Dr. F. W. Robbins, Detroit—I think the paper is written in a strong style and will impress the readers. I think that mental impression is of more importance to the youth than the mere knowledge. It seems to me that the period of life at which the intelligent young are instructed in these matters

is, in most respects, too late. There are two periods in the boy's life when he should be impressed with this knowledge: 1. The early period, which comes at the ninth or twelfth year. Children at this period can scarcely be expected to be much impressed with such a paper in which there is so much discussion. Then it is that the boy more particularly requires the attention of the father and not of the physician. 2. At a later period, when the boy goes to school, he should be given this knowledge. I believe it is an injustice to allow these boys to go without this precious knowledge. I think it is not so much the knowledge as the mental impression which keeps these boys moral.

Dr. DeLancey Rochester, Buffalo—I am particularly interested in Dr. V.'s paper because twelve or fifteen years ago I presented a paper on a similar subject before the American Academy of Medicine. I believe the proper time for giving such instruction to these boys is during the high-school period, and the best way of imparting this knowledge is by the proper teaching of physiology in the schools by physicians. The whole subject of physiology should be taught and not—as at present—only in part, and that imperfectly. The truth should be taught; and that is that there are two great functions of the organism, namely, the preservation of the individual and the perpetuation of the race; that the functions of the various organs of the body are subsiduary to one or other of these great functions.

A thoroughly careful and competent teacher could show by the history of the complete separation of the sexes in plant and animal life that there has been developed the most beautiful in flowers and the noblest in animals. Time does not permit me to elaborate the scheme, but I thoroughly believe that physiology taught in this way would arouse in each one a noble respect for the human body in all its parts and a determination to do all in his power to develop a perfect human being and to keep it pure and holy. I believe that by careful selection of teachers physiology could thus be taught in the public schools.

Dr. Theodore Potter, Indianapolis—My experience has led me to somewhat the same conclusions as Dr. V. has reached There are two periods in the boy's life when such instruction can be given: 1. At the time of puberty, when the boy is at home under the influence and control of his parents and family physician; 2, when he goes to school or college. At this latter period he will look on such instruction as may be given him in a serious way. If I am allowed to differ with the last gentleman who spoke, I must say that I question whether the public school or the intermediate school will give the proper opportunity for giving such instruction, because the boy is more or less immature at that time and looks on such teaching as frivolous and makes a joke of it. Later he will

listen and take it in a more serious way. When attempts have been made by public school teachers to instruct the boys in this country they take it as a sort of joke; but this is not so among the college students. It really is surprising what a great amount of ignorance is displayed by those young men who are entering or who have gone through college. I have had experiences in the west as well as the east in institutions, and I have seen the most lamentable examples of young men, not bad men at all, who have gotten into trouble largely through ignorance. This is the point: The young man goes free for the first time when he enters college and, since human nature is imperfect, when he reaches this time of freedom something goes wrong and it is largely through lack of proper instruction. I believe Dr. V. is right when he states that when the young men attend college, reaching the age of maturity, then is the time that serious instruction should be given.

It has been my habit, and this I think should be done by the physicians all over the country, to say to my patrons who have boys, that I am going to talk to a few boys at my house and if they wish they may send their sons. Invariably I have met with a willing response, and I know that my efforts have been crowned with success, so far as instructing them is concerned.

Dr. M. M. Smith, Austin, Texas—I desire to lay special stress on the influence exerted by the home circle in the prevention of venereal diseases in young men. During the earlier years of the boy's life he is constantly with the mother and if she is careful of his conduct and associates and watches him carefully and teaches him obedience and morality during these years, he will show the value of this watchfulness in his character. Equally important is the duty of the father, and especially is this true when the boy reaches the age of puberty. This is the time the father should be his companion and friend, and especially watch his conduct and companions and guide him safely over these few years when he is developing into complete manhood. It is too often the case that the parents are earnestly at work to further some charitable and noble enterprise in that public-spirited manner; but at the same time they are neglecting their own children at a time when their influence will leave its effects all through their lives. The subject should engage the attention of the parent, the teacher and the physician; in fact, on the proper solution of the subject depends the future welfare of the nation.

Dr. Frank A. Jones, Memphis, Tenn.—I doubt very much the propriety of bringing this matter before the laity as advocated. By doing so there will be a tendency for the boy of young age, who is disposed to look further into such matters, to investigate further with disastrous results. We meet sexual neurasthenia which is purely an imaginative condition;

many come to us with trivial affections and even talk of suicide, and this may be the result of publicity being given to such matters. I do not think this subject should be placed before the public. Try and enjoin on the young men this, that the way of the transgressor is hard. I think the young men can be reached better through the medical profession than through the public press or other means of publicity. I think it would be wise to distribute Dr. V.'s article by the thousands to various physicians in the American Medical Association.

Dr. E. L. Stephens, Des Moines—Before the boy reaches the age of puberty I think he should be taught the sacredness and naturalness of the sexual instinct, and this can be done in a quiet way. There has been published a book, "Almost Fourteen," by Mortimer Warren, which I think is most suitable to place in the hands of young men, and the only one that I have been able to find after nearly forty years' search. That is, I believe, the only one that can be safely placed in the hands of young men as being scientific and pure. Since the American Medical Association has been reorganized we now have a means whereby we can legitimately place such information before the public; previously it could only be done by quacks. Now each councilor in each district has the privilege of using the public press for disseminating proper information. We can present through the public press the right ideas and the right information which would offset the wrong impressions given the laity by charlatans and quacks.

Dr. Walter Shropshire, Yoakum, Texas—I think that if anything is to be accomplished toward protecting the young of this country it should be done at a time when it would be effective. When the boy reaches college it then is too late. If we should wait until some definite period we should select that period when such instruction would be most efficient and that is concomitant with the development of the sexual function in the boy. When he first learns of this function he should be taught the dangers of its misuse. I really believe that physicians should teach physiology in the public schools and, at the same time, they should teach regarding the sexual function and the dangers which its abuse entail.

Dr. Ferd. C. Valentine, New York—Naturally it was impossible in the brief synopsis which I presented to cover the entire ground; that has been largely done by the gentlemen who so kindly discussed my effort.

With regard to Dr. Walsh's remarks, I may say that the universities in Germany, beginning in Berlin with Lassar, have given instructions to students regarding the dangers, etc., of venereal diseases. Lectures are delivered to students of all the departments of the university. The brilliant success of Lassar was so encouraging that it caused an increase in the number of such lectures, with exceedingly good results. But

you may ask how many of those young men who came there to be educated and were instructed in venereal diseases had not unfortunately educated themselves. In Berlin the war is directed against the repetition of contracting such diseases, as well as instructing regarding their nature and results. I agree with Dr. Walsh, that some method should be introduced in this country to teach the young men the evils of venereal diseases. I wish to emphasize his remarks and say that university students who attend these lectures derive much benefit at the time when they certainly are qualified to receive such instruction.

What Dr. Robbins says regarding the time for receiving such instruction is undoubtedly correct, but he says it is not so much knowledge but impression which is of value, and that they should be taught at an impressionable age. But what is that age? That varies with the individual. There are some boys who are impressionable at the age of 12. I am now treating one not quite 14 years old, who has a gonorrheal prostatitis. This boy is the only son, in a good family, and has been correctly raised. With six others he was solicited by a girl of 14; all six contracted gonorrhea. Had he been instructed at the period when he was of the impressionable age his misfortune might have been averted.

CONTINUED FEVER—NEITHER MALARIAL NOR TYPHOID.

T. J. HAPPEL, A.M., M.D.
TRENTON, TENN.

It may seem that the subject of continued fever is becoming too common in our meetings to merit any further time or discussion, but, so long as we are met with the *ipse dixit* of some bookmaker, that there are but two types of continued fevers, and that all such fevers must be classed with one or the other, in fact must be either malarial or typhoid, then those of us who do not accept any such dogmatic statements as necessarily true, have a right to give our reasons for our dissent. Our medical brethren in the north, as a rule, contend that every continued fever is either typhoid or malarial, while in the south many of us are sure that we have a third fever, which merits a careful study, the mortality of which is so slight that opportunities for postmortem study have been almost *nil*.

The blood and secretions have been examined microscopically and chemically so far with negative results. A sufficient number of examinations have been made of the blood to show that it does not respond to Widal's test, nor does it show the presence of the *Plasmodium malariæ*. In none of these cases has the estivo-autumnal parasite or the *Bacillus typhosus* been discovered, yet we are told by the bookmakers that the technic was faulty; that the examinations were improperly made. While not prepared and not competent to make any of these examinations, or to apply properly any of the

tests in these cases, I have too much confidence in the ability and honesty of the laboratory workers who have made these reports to doubt their correctness. (Just at this point I would offer this suggestion, that no specimen sent to a microscopist or chemist should be accompanied by any clinical history of the case, to avoid the warping of the judgment of the examiner. He should be requested to examine the specimens sent for the evidences of malarial or typhoid fevers, and to report the result of his examination.) We can more often find what we look for than the unknown or unexpected; hence, I say that the clinical history of the case should be withheld.

The teaching of the present day, as pointed out by the learned Dr. Jacobi in an address at Saratoga before one of the Sections of the American Medical Association, in which he spoke with no uncertain sound, does not develop the skilled clinician. The bedside study of diseases has been relegated to the background and the student is impressed with the idea that the microscope will tell him positively all about his case. In many of our diseases the microscope is as yet powerless. One laboratory worker reports and describes a specific germ for a disease, and another soon after reports one entirely different.

In this fever to which I invite your attention the microscope fails to show the *Bacillus typhosus* or the *Plasmodium malariæ*. Reference was made in a former paper to the fact that a number of skilled laboratory workers had investigated the secretions and blood, both microscopically and chemically; by one in more than one hundred cases, and by another in thirty-eight cases; with the same result in each, both asserting positively the absence of anything distinctive of either typhoid or malarial fevers. In numerous other instances the same results have been obtained by others. Yet we are told that Dock of Ann Arbor has proven conclusively that the cases of so-called continued malarial fever in

Texas were cases of true typhoid fever, and Dr. Osler asserts that he feels sure that all the continued fevers except those of thermic origin will yet be divided into but two classes, malarial and typhoid, which position is equivalent to asserting that our microscopists who have reported otherwise were not qualified to properly investigate the etiology of these fevers and that our chemists had not skill enough to apply the tests necessary in that part of the laboratory work. His argument is along this line:

Typhoid fever in Baltimore and Philadelphia, where malaria exists, is not essentially different from the same fever in Montreal where no malaria is found; hence the other continued fevers in the south where malaria exists and where typhoid fever is rarely found must be true malarial fever.

This can not be said to be a first-class argument. A fever that is totally distinct in every respect from the malarial or typhoid fevers except one, viz., duration, can not of necessity be one of the two types. Not many years ago we recognized only two types of the eruptive fevers: measles and scarlet fever; yet recently measles has been divided into rubeola and rötheln, and roseola is certainly distinct from either, and within the last two years an effort is being made to divide scarlatina, as it had been known, into two distinct diseases, each having its own *causa causans*.

Our boards of health have not yet accepted the doctrine of a disease resembling variola, pseudo-smallpox, having the same relation to variola that rötheln does to rubeola, and the new differential of scarlet fever does to true scarlatina. This effort on the part of those standing high in the councils of medicine to decry anything new not originating with themselves does not tend to encourage individual research. The impetus to medical and surgical investigation along many lines has often been given by the country practitioner or surgeon, as evidenced in the cases of Marion Sims of Alabama and McDowell of Kentucky.

I lay no claims to the discovery of a new fever, but

I do take the position that we have here in the south a continued fever that is neither typhoid nor malarial.

In a former paper, I referred to a number of such cases treated by me in the last ten years, and since that paper was written a number of others have been under my care, and very favorable opportunities have been seized on to study these cases at the bedside. The conclusions have not been verified by any autopsies as all of them have recovered. Since July, 1902, I have treated six cases (a total of about one hundred cases), lasting three weeks in the shortest to five in the longest. In each one of these cases the diagnostic points were strongly in evidence. The first case was ushered in by a cold stage, not the chill of malarial fever, but rather the algid stage of la grippe followed by a slight rise of fever, and on the following day at about the same hour more rigors with intense headache and a temperature of 104. The parents, supposing they had an intermittent fever with which to deal, had given calomel, followed by quinin in full doses until the daughter was cinchonized, as evidenced by tinnitus aurium, and deafness. When called on the fourth day, I found a normal pulse, profuse perspiration and, while the parents thought her free from fever, a temperature of above 104, no vomiting, no special complaint except headache. Concluding that I had a malarial attack to deal with, I increased the number and size of the doses of quinin sulphate so as to give between 25 and 30 grains before the time for the next paroxysm on the following day, with directions to continue until my return in 48 hours. My next visit showed no effect from the quinin except to increase the deafness and to disorder the stomach and destroy the appetite. I found pulse 78, temperature 104, no tympany, no engorgement of the liver or spleen, no broad flabby tongue, no jaundice, no special complaint about anything except headache. I abandoned the further use of quinin, and prescribed salicylate of soda and carb. guaiacol as follows:

℞. Sod. salicylat. .....ʒiii
Guaiacol carb. .....ʒi
Milk of magnesia .....℥ii
M. et ft. Mist. S. shake well. Teaspoonful every four hours.

A mixture of aconite and gelsemium was prepared to aid in reducing temperature, and for the same purpose frequent sponging with tepid water was directed. On my next visit there had been two cold stages in each 24 hours, but the temperature had not risen above 103. The treatment as outlined above included free and frequent spongings with tepid or cold water, as preferred by the patient, and when the temperature resisted these measures and persisted in remaining up to 103 degrees, 5 grains of acetanilid to reduce, but not to be used if it could be avoided. Only 9 grains of quinin sulph. each day were given until the case fully recovered, which required four weeks from date of going to bed. In this case there were two well-marked rises of temperature in each 24 hours. There was no anorexia, and not a single point of resemblance in the case to typhoid fever.

In one of my other cases the attack was ushered in, or, rather, preceded by an attack of irritation of the alimentary canal, amounting to almost a case of enteritis, caused by the immoderate and excessive drinking of ice-cold lemonade at a Sunday-school picnic. For several days all symptoms were masked by the frequent small, griping, mucous discharges from the bowels, the excessive thirst, tympanitic distension, and an inability to retain anything taken per os. As these symptoms subsided, leaving the whole abdomen tender, the tongue, which had been red and small and long, returned to its normal state, the thirst became less, and the rises in temperature, in spite of all that could be done, were three in 24 hours. Quinin per rectum, after a failure to prevent these exacerbations per os, had no effect on the fever. In this case I was not content to use one form of quinin alone, the sulphate, but resorted to the bi-sulphate, and later to the salicylate.

At the end of ten days, no impression having been made on the course of the disease by the antimalarial remedies used, they were abandoned, and the guaiacol-carbonate and soda salicylate of the first case substituted, and under this course the patient was carried through a sickness lasting five weeks, to be followed in two weeks by a relapse from imprudent eating. This lasted two weeks.

The third case lasted four weeks, receiving thorough antimalarial treatment for one week, which was abandoned as useless, and the medicines referred to in Cases 1 and 2 substituted, supplemented later on by a mixture of tr. iodin and Fowler's solution every four hours. The temperature chart showed four rises of temperature in each 24 hours, and later on three, then two, and then one. In this case, from the effect of quinin used during the first week, the digestive organs were weakened so that only malted milk could be retained for a few days, though a desire for food was ever present, and the patient, a female, aged 10 or 12 years, never failed to report herself feeling better, and when her temperature would rise to 103 or 104, the pulse rate was never higher than 90. There was never a dry, harsh skin, never bilious vomiting, never nausea except that which resulted from the use of the quinin the first week. She was always ready to eat, but never thirsty. All the secretions were normal, the bowels generally acting once every 24 hours. At one time she was allowed by her mother to eat sardines, with no unpleasant sequences. At the end of the fourth week the fever ceased, terminating, as shown by the chart, by lysis. In this case there were no profuse sweats as there were in the first and second cases.

Case 4 was a boy, aged 8 years, just on the opposite side of the street from Case 3. It was very mild, showing only two rises of temperature in 24 hours. This patient was saturated to deafness with quinin, which was then laid aside except 6 grains per day, and the guaiacol-carbonate and soda mixture alternated with

the iodin and Fowler's solution used throughout the case. On the twenty-first day the fever terminated by lysis. This patient was hungry from the time he was put to bed until he was dismissed. At no time was there any derangement of any of his secretions. With his light diet he became constipated and was given cascara two or three times each week. During the second week he was given four doses of calomel, gr. 1/10.

Case 5 was a repetition in every respect of Case 4. These cases are added to a long list of similar cases already treated by me, with only one death, due to congestion resulting from a disobedience of directions. While a few of the cases have lasted six weeks the rule is recovery in from three to four weeks. We do not expect, except in very young children, recovery before the end of the third week, and my invariable answer to questions of the probable duration of the disease and the prognosis is that they will recover, but not before the expiration of 21 days from the date of going to bed.

In my last case treated (November), the temperature, after dropping the quinin, slowly but steadily decreased each day, until on the eleventh day from going to bed it dropped to normal and did not rise above 100. This case appeared to be a duplicate of Case 5. But the slight rises of temperature alone and the little difference between the daily maximum and minimum point is no indication of the duration of the attack, several of such cases having lasted from four to six weeks, and in one case especially referred to in one of my former papers on this fever, after continuing for three weeks with a maximum temperature of 101 to 102, during the fourth and fifth weeks there were daily ranges from normal to 104 and over. These cases are the ones we expect to terminate by crisis with profuse sweats.

The cases of this season have demonstrated the fact most conclusively that quinin will not cure this type of fever. I have used it in varying doses per os and per rectum. In none of these cases was the use of it dis-

continued until decided deafness was produced, no attention being paid to any of the other symptoms of cinchonism. I have not used any of the quinin salts in from 20 to 40 gr. doses, never do, but have repeated in from 3 to 10 gr. doses until the effect was self-evident, and then have maintained this effect for from three days to a week and in some cases to ten days.

Reference has already been made to the use of microscope and chemical investigations in these cases, hence I will devote no further time to this branch of the subject, not being able to speak *ex cathedra* myself in the matter. I desire, however, to emphasize the point of difference from a clinical standpoint, and permit me to add that where the bedside study of a case teaches plainly one thing and the work of the laboratory another, I, for one, would strongly incline to the teachings of the sick chamber. It has been demonstrated time and again that the laboratory worker has agreed with the clinician in this fever, and yet the question of competency has been raised; while no question is raised in the case of other workers in the laboratory when they have discovered, or thought they have found the micro-organism for which they were looking. These cases have not, then, been ushered in, as a rule, abruptly by a chill, but have had usually for a few days or a week prodromes of malaise, then rigors, sometimes a slight chill, followed by a slight rise in temperature, which does not generally attain its maximum for from three days to a week. While this fever presents marked remissions, for the first week the remissions are not so decided in the second week, but are more so in the last week, amounting to intermissions with profuse sweatings. The chills, or cold stages, are accompanied by aching of sometimes one set of muscles and sometimes in all, resembling in that respect la grippe. After a day or two all pain ceases, and the patient complains of nothing. The remissions in this type of fever vary from one to four in 24 hours, and may be made more

marked by sponging with tepid water in all cases, by aconite and gelsemium in a few, and by the coal-tar derivations in almost all of them. As a rule, sponging produces a condition of euphony and the patient never objects to its use.

There is no nausea and vomiting; never bilious vomiting, no jaundice, no clay-colored stools, no diarrhea, and, if any departure from the normal in this respect, constipation; no tympanites, unless it be due to some imprudence in eating; no tenderness over the abdomen; no rose-colored spots; no marked enlargement of the spleen or liver, unless there has been a history of malaria preceding; no anorexia, but, on the contrary, in many cases a desire for food all through the sickness; no broad, dry, flabby tongue, with its yellow pasty coating, but, as a rule, a tongue nearly normal in every respect, and when coated it is thin, of a light color. We have, in none of these cases, the pointed, narrow, dry, fissured tongue, with its red edges and dry, coated, and frequently cracked center. The tongue never bleeds, in fact the rule is all typhoid and malarial appearances are absent from the buccal cavities and the rest of the alimentary canal. The digestion is rarely impaired for food suitable for the sick, though I have noted abdominal distension when solid foods have been administered or allowed. There is no mental hebetude; in fact, generally the brain is not affected at all and ordinary business matters could be attended to by many of these patients with their usual business acumen. We never have any low muttering delirium as in typhoid fever, nor the active symptoms of malarial fever. The facies of the patient is normal, though he may be thin from loss of flesh attendant on the long drawn out fever. The muscular strength is not impaired as in typhoid fever, but its loss is in proportion to the length of the attack and heighth of temperature. In one patient sick six weeks, with a temperature reaching 105 daily for nearly two weeks, the loss of weight was slight, but

the muscular tone was much impaired but was rapidly regained.

That these cases are probably toxenteric, as claimed by Dr. J. A. Witherspoon of Nashville, is evidenced by the fact that when relapses have occurred, in my practice they could be traced to imprudent eating. In no case of the many treated have I ever seen any tendency to epistaxis, though in some persons who were predisposed to tamper with their nasal organs, a few traces of blood have been noted in the secretions blown from the nose. The kidneys have acted, as a rule, normally, the quantity of urine being diminished where the skin has acted too freely. We have no dry harsh skin in this fever as in typhoid fever, nor is sweating any evidence as in malarial fever of a decrease in temperature, the skin in this fever being frequently bathed in perspiration when the temperature is at its maximum.

One of the most marked peculiarities of this fever is the disproportion between the pulse rate and the temperature. The ratio is frequently inverse, the pulse beating at from normal to 90, with a temperature of 103 to 105 or even higher, and the same may be said to be true in regard to the temperature of any exposed portion of the surface of the body. The hand applied to the pulse and then to the forehead would lead anyone not using a thermometer to say that there was a perfect freedom from fever. This condition of things has been reported to me time and again, and yet when the thermometer was used in the mouth or axilla a high temperature was clearly shown. The hand applied over the bowels or to the back under the clothes and cover frequently shows fever. In these cases, no proper nursing can be done without the use of the thermometer every two hours, and a record is required to be kept in every case, with, however, a verbal direction not to disturb the patient when soundly sleeping. I might refer to the fact that it has been my experience that the nearer a direct proportion between pulse and temperature, the

more confidently is a termination by lysis to be expected at the expiration of 21 days, and vice versa. In those cases of indirect ratios the termination is more likely to be by crisis and profuse sweating. In this class of cases, when the fever begins to intermit, strychnin is demanded. I might consume time indefinitely on the subject, but my time is up.

TREATMENT.

As to treatment, a few words. As soon as satisfied that the cases are not malarial, which should be determined in four days, quinin should be abandoned or used as a tonic in 3 gr. doses. I have obtained my best results by alternating the following prescriptions for an adult:

℞. Guaiacol carb. ..... ʒii
Sod. salicyl. ..... ʒvi
Bismuth. lact. ..... ℥iv

M. Sig.: Take one teaspoonful every four hours.

℞. Tr. iodin. ..... ʒss
Liq. potas. arsenit. ..... ʒi
Aquæ ..... ℥iv

M. Sig.: Shake. Teaspoonful every four hours. Alternate with the first mixture.

All symptoms are met as they arise, but unless some idiosyncrasy is manifested by the patient and the temperature runs high, these two prescriptions are kept up regularly night and day, but where the grade of fever is low they are usually omitted at bed time, say 10 p. m. I have used many intestinal antiseptics in treating this fever, but have had the best results this year from the above course.

The temperature is reduced by the free use of tepid or cold water, allowing the patient to choose.

The diet is liquid or semisolid, buttermilk being preferred to anything else when the patient can use it.

SOUTHERN FEVERS.

WILLIAM KRAUSS, PH.G., M.D.
MEMPHIS, TENN.

In assigning this subject to me, our secretary said he wanted to show that some missionary work on the subject of fevers in the Mississippi Valley was being done, despite the controversies one sees about slow, continued, typho-malarial, and other fevers. With this idea of the scope of the paper, fevers of sepsis, tuberculosis, rheumatism, syphilis, etc., have not been given a place.

The cases presented are to be looked on as types of a number of cases seen by the author, and are believed to serve the purpose better than an elaborate discussion for which we have no time. None of the histories are complete, but enough is offered to make the point intended.

1, ACUTE ESTIVAL FEVERS; 2, CASES WITH AUTOPSIES.

CASE 1.—W. B., male, white, aged 54, was brought to St. Joseph's Hospital in July, 1900, in an unconscious condition. From others it was learned that he was a passenger on a train coming to Memphis, and was suddenly taken with what was described as "congestion"—a term very much in vogue to denominate sudden fevers usually terminating in death. His temperature on admission two hours later was 108 F. The notes of the attending physician state that ice baths, subcutaneous injections of antipyrin and quinin had failed to bring about a change, and he died in six hours without ever regaining consciousness.

A blood examination had not been ordered, but the antemortem diagnosis read "pernicious malarial fever." The following from the necropsy report is of interest:

Autopsy five hours after death; rigor mortis; body of a very plethoric man; capillary stasis; ecchymosis; incision over calvarium bleeds freely; excessive hyperemia of encephalon; blood smears taken. Abdominal viscera pale, no marked changes; pleuræ free, lungs hyperemic; heart very firm, right ventricle full of dark fluid blood; smears from this also; no plasmodia in either. Postmortem diagnosis, sunstroke.

CASE 2.—Colored man, aged about 45; arrived at city hospital comatose in August, 1900. Attendant's notes showed rectal temperature of 107 F. on admission; plasmodia in blood. Was packed in ice, quinin and saline infusion subcutaneously; slight reaction; death next morning.

Autopsy: Marked hyperemia of all the organs; heart contains thrombi which are nearly white.

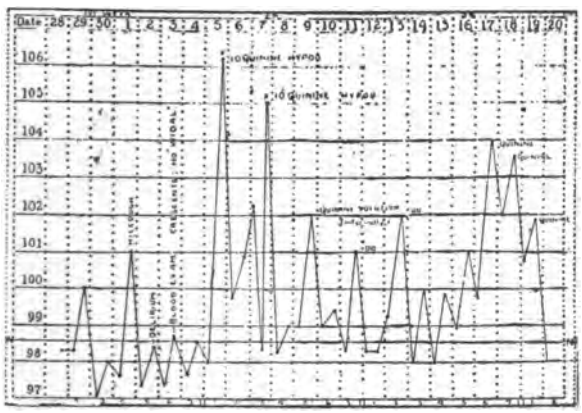


Chart 1. case 3.

The tissues and blood were taken to author for microscopic examination.

Smears from lungs and brain contained estivo-autumnal parasites, no crescents; splenic blood not fit for examination. Microscopic sections showed marked pigmentation in liver, spleen, kidneys and lungs. Pigment is siderin, not melanin. Very marked fibrous endarteritis, especially of the coronaries, doubtless syphilitic. Tissues had been in formalin very long and efforts to stain parasites in the sections proved unsatisfactory.

These two cases are reported as different comatose fevers with very similar symptoms. If the attendant of the first case had made a blood examination he would not have fallen into the error of giving quinin to a patient with sunstroke—it was a serious mistake.

II. CONTINUED ESTIVAL FEVERS WITH DIARRHEA, 2 CASES, WITH ONE AUTOPSY.

CASE 3.—J. M., aged 26. Admitted to St. Joseph's Hospital Sept. 28, 1899. Had been sick at Little Rock, Ark., with dysentery for a week; had had vomiting and daily chills. On admission showed abdominal tenderness, slight tympany, gurgling in right iliac fossa; spleen enlarged and tender; tongue dry, red and hard, with fissures; low delirium at times. The accompanying temperature chart completes the history, but the record for the first five days is not correct, as it was found afterward that he had nocturnal chills and exhibited much prostration in the mornings. A physician in consultation insisted on its being typhoid. The blood analysis appears on the fever chart; he had the malarial leucocyte count, estivo-autumnal parasites, crescents, no Widal reac tion. The treatment was antimalarial, salines for the dysentery. He was discharged cured at the end of four weeks. His paroxysms being nocturnal, they had been overlooked and the accompanying dysentery helped to produce the typhoid symptoms. Heroic quininization changed the picture.

The temperature chart and blood examination saved this case from being classed as typhoid fever, and death would have been the result.

CASE 4.—(Record taken from the Reports of the Marine Hospital at Memphis.) The writer saw the patient several times and assisted at the autopsy:

J. F., colored; native of Louisiana; admitted to the United States Marine Hospital at Memphis Dec. 26, 1896, and died Jan. 3, 1897.

History.—Patient was a well-developed and healthy-looking colored boy. He had been sick for four days with chills and fever. Initial chill came on in the evening and was very severe; had a similar chill every evening up to day of admission. His bowels were very loose from the first. Discharge watery and preceded by pains in the abdomen. Temperature on admission was 40.5 C. Pulse poor; tongue round and flabby, clean and red on edges and moderately furred in center. Neither liver nor spleen noticeably enlarged. Was given tincture digitalis on admission and was ordered quinæ every four hours with bismuth and mixture for diarrhea. His temperature on the morning of the 27th was 39 C. and pulse 90; bowels loose but less so than on preceding day. His blood showed a large number of very small extra-corpuscular "rosettes" and some granular bodies. The segmentation in rosettes was not as distinct as usual. On admission the diagnosis of intermittent malarial fever was made to conform to official nomenclature, but the blood examination showed at

once that the disease was estivo-autumnal, or, as the nomenclature has it, remittent fever. The possibility of a mixed infection was considered but abandoned. There was an entire absence of tympanites, gurgling and iliac tenderness. The stools were more watery and paler than typhoid stools, but still quite suggestive of typhoid. While the malarial nature of the case was clearly established, still the fever was a hard one to classify as to the kind of malarial fever present. The history from onset to admission pointed to a double tertian or to the variety of estivo-autumnal fever marked by quotidian paroxysms.

The appearance of the rosettes and the temperature curve for the first four days indicated the quartan type of estivo-autumnal fever, while the subsequent temperature curve showed regular tertian remissions. His diarrhea yielded slowly to treatment, declining by the 31st to two passages a day. On the 30th his blood showed both intra- and extra-

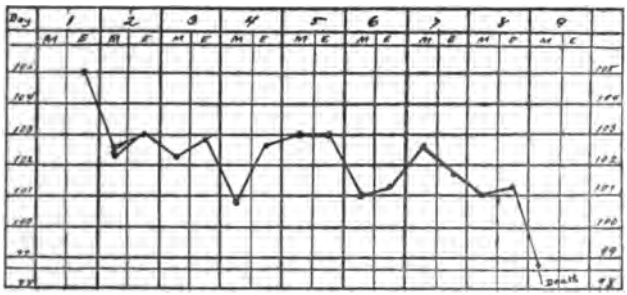


Chart 2, Case 4.—J. F., U. S. M. H., Memphis. Temperature transposed to F. scale. Remittent fever, with diarrhea, perforation and death. Prodromes: Chills daily, 4 days; one on admission; parasites in blood.

corpuscular, small hyaline bodies, and a few large and actively motile pigment grains or blocks—two or three to each body. The next day only a few extra-corpuscular pigmented bodies could be found. His mental state from about the third day became rather foggy—a fairly typical typhoid state in fact.

On the 1st of January, the sixth day from admission and the tenth since onset of disease, he had several severe attacks of colic, and these recurred on the 2d. On the night of the 2d his pulse failed rapidly, tympanites developed, and it became evident that he had sustained an intestinal perforation. He died at 10 o'clock on the morning of the 3d, just twelve days from date of first attack.

Necropsy, twenty-four hours after death. Body fairly well nourished. No scars. Calvarium not opened. Lungs normal except for slight congestion. Pericardium contains about 200 c.c. of fluid. Heart rather small, but otherwise normal. Liver,

spleen and kidneys normal in appearance and size. Intestines showed general fibrinous adhesions. About 250 c.c. of fluid present in abdomen. The ileum presented a large number of follicular ulcerations. These ulcerations were all opposite to the attachment of the mesentery. Peyer's patches were not at all involved. The mesenteric glands were not enlarged. Two perforations were present—one 4 cm. from the ileocecal valve and the other 12 cm. There were several ulcers, in which the coats were eroded down to the peritoneum. At about 20 cm. above the ileocecal valve there was an area about 2 by 4 cm. where the entire mucous membrane was eroded. Sections taken through the ulcers showed a typical follicular ulcerative process and an entire absence of any of the microscopic appearances of characteristic typhoidal ulcers.

The ulcers were small, funnel-shaped, less than 1 mm. in diameter, with elevated edges; the perforations were about 2 mm. in diameter and closed by fibrinous exudate. Dr. Young and I discussed the Widal test, but this was in its infancy; neither of us had any cultures of typhoid on hand, and Johnston's dried blood method was as yet unknown; typhoid fever is, of course, not scientifically excluded. We both wanted to believe it was typhoid fever; it was between Christmas and New Year, but the blood and postmortem evidences were against such a diagnosis, and it was reported as a case of remittent fever.

III. CONTINUED FEVERS WITH CONSTIPATION, 2 CASES.

During my term of service in the summer of 1899 at St. Joseph's Hospital I had 248 cases of fever, and of this whole number there were only 3 in which there was a question of diagnosis; one of these is reported above. During 1902 I was interested in 2 cases, which follow:

CASE 5.—O. G., aged 19, male, drug clerk; had had intermittent fever a year ago; malaise this time for several days, followed by catarrhal bronchitis. Seen first by Dr. Baldwin (to whom I am indebted for this history) on Oct. 26, 1902. Provisional diagnosis, la grippe; salines and salicylates administered; remission on second day, rise on third; cinchonized for three days. Temperature during first week, morning 100 to 101.5 F., evening 101 to 102, pulse 80 to 85. Dried blood spreads brought to me November 2 showed intracorpuscular estivo-autumnal parasites, about half grown, no crescents;

infected cells generally crenated and small. Leucocyte count of 300 cells: small lymphocytes, 9.3 per cent.; large lymphocytes, 30.3 per cent; polymorphonuclear, 58 per cent.; eosinophiles, 1.3 per cent.; basophiles, 1 per cent.; a few microcytes and small nucleated forms, phantom cells and polychromatophiles. No Widal reaction in any dilution. He was thoroughly quininized again without visible change, and after another week malarial infection was still present; no Widal; small lymphocytes, 20; large lymphocytes, 14; polymorphonuclears, 64; eosinophiles and basophiles, each 1 per cent.

Pulse still 85 and good volume, now slightly dicrotic. Temperature runs from 100 to 103 without change in pulse. This time he was given 90 grains quinin hydrobromate in 24 hours; it seemed to have the effect of running up the fever, for he had 102 in the morning and 104 in the afternoon following, and for four hours there was intestinal hemorrhage, at first slightly clotted, then fluid and carmine red; there were no piles. Cold sponging was followed by a fall in fever and profuse diaphoresis. The fever now gradually declined, reaching normal on the 32d day of the disease.

There was marked tendency to constipation throughout, combated by salines, and when these were suspended some tympanites would develop. Nose bleed once in the first week; a few petechiæ present once, but rather persistent; subsultus once, early; mental hebetude at times. Entire course mild and marked by very slow pulse. Widal reaction constantly absent.

Case 6.—This patient took to his bed June 14, 1902, and was removed to a private room at St. Joseph's Hospital on the 16th. The previous history was that of malaise for a week, with gradually rising fever. The attending physician found estivo-autumnal organisms and pigmented leucocytes, and gave several intragluteal injections of quinin bimuriate with urea, 0.5 gram each in 10 c.c. of water at six-hour intervals. There was albuminuria with casts and gradually aggravating oliguria during the first week. Renal and frontal pains were constant and troublesome symptoms. There was obstinate constipation and intestinal paresis throughout. The patient could not bear cold to the surface, even icebags produced local neuralgia, and the temperature was reduced with thermol when it got near 104; for this reason the fever chart has no value.

At the end of the first week in bed, no blood evidences of malaria being present and fever still persisting, several trials with the Widal test were made, but without result. The diagnosis of paratyphoid was suggested by one consultant, but no cultures were obtainable. The fever definitely subsided at the end of three weeks without further resort to quinin.

This patient was seen by three very competent prac-

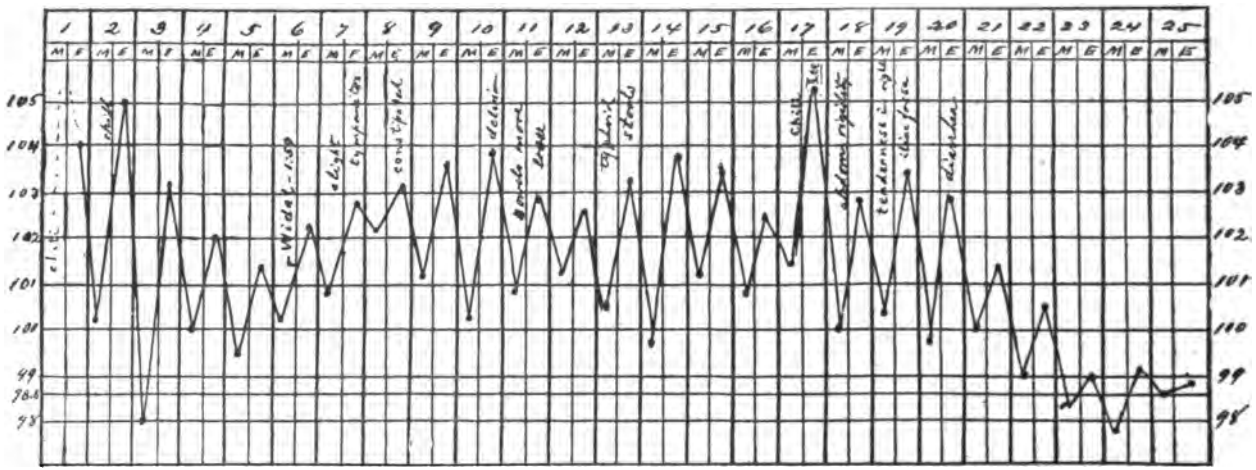


Chart 3, Case 8.—W. J. R. Typhoid fever, with chills. Blood, first day: No plasmodia; no pigmented leukocytes; white cells, 12,020, stained eosin and m. blue; small lymphos., 22, or 11 per cent.; large lymphos. and basophiles, 46, or 23 per cent.; polys., 130, or 65 per cent.; eosinophiles, 2, or 1 per cent. Sixth day: No plasmodia; Widal in 1:50=30 min.; small lymphos., 18 per cent.; large lymphos., 15 per cent.; polys., 67 per cent.; eosinophiles, 0 per cent.; total leukocytes, 9,340. Seventeenth day: No plasmodia; white cells, 16,310; 77 per cent. polys. This case presents a leukocyte proportion resembling that of malaria, except as to polymorphonuclear cells.

titioners with all the resources of modern medicine at their command, yet one positively asserts that it was a case of malarial fever and proves it by his slides; the second is equally as certain that it was typhoid fever, despite the failure of the Widal test, and the third, who is our honored secretary, modestly suggested that it was paratyphoid. The writer's own opinion is not worth much, since he was the patient. A very complete history was taken, but it throws no light on the case.

During the summer of 1900, 26 cases with history of acute malarial fever were admitted to my service at St. Joseph's Hospital, none of which received any antiperiodic treatment after admission. The blood was examined in 11 without showing infection. They were held to await further recrudescence of the paroxysms, but the fever gradually subsided. They were booked as postmalarial malaise. Here is the history of one:

CASE 7.—L. F., female, aged 24, white, married. Lived in St. Francis bottom; admitted June 10, 1900, with history of tertian chills. Temperature on afternoon of day of admission 102 F., pulse 110 and small. Liver and spleen enlarged. Blood analysis next morning: reds, 3,640,100; hemoglobin, 70 per cent., indicating reduced color index; whites, 6,470, differential count of 200 (eosin and methylene blue). Small mononuclears 10.5 per cent.; large mononuclears and basophiles, 28.5 per cent.; neutrophiles, 54 per cent.; eosinophiles, 8 per cent. No malarial organisms; some leucocytes pigmented.

Blood count five days later showed 3,981,000 reds; hemoglobin, 80 per cent.; whites, 10,180, 70 per cent. of which were neutrophiles; eosinophiles and small lymphocytes gaining. Discharged on June 20 feeling quite well.

The following is a case of typhoid fever with irregular temperature curve:

CASE 8.—W. J. R., aged 21, male, white, clerk. After a two-day malaise he went to bed on July 20, 1901, with a chill, followed by high fever and going off in a sweat. The first temperature reading, taken at this stage, was 104 F. Blood examinations on fever chart; malarial count, leucocytosis, no plasmodia. Next day he had another chill, followed by rise of temperature to 105.2; no plasmodia; no local symptoms to account for chills, pus suspected; bowels constipated and paretic; enema and salines given. Fever then assumed a remittent type and leucocytes diminished; relative increase

BOSTON MEDICAL LIBRARY MAR 16 1928

of small lymphocytes. Widal reaction positive on first and only trial, on sixth day. The case now ran typically typhoid until in the third week there was another chill with leucocytosis; no plasmodia; no quinin given. From now on fever gradually subsided and he made an uneventful recovery. A year later he developed a suppurative appendicitis and was successfully operated on. I am now quite sure that this is what complicated his typhoid fever. I should add that no adhesions were found at the operation. The accompanying temperature chart completes the history.

Without a blood examination this would have been regarded as a case of malarial fever with intercurrent typhoid.

Now, in conclusion, a few words as to our methods of diagnosis. The Widal reaction is open to fallacies. Even if the culture is just right and the drop contains no fibrin, we have errors estimated at from 3 to 12 per cent. either way. Frequently a single test is all that can be made and the reaction, if transient, may escape us. We also know that it may persist from a former attack and which was perhaps not recognized.

On the other hand, to what do we refer the Widal test as standard? When can we say that it has failed? The blood culture test only appears in about 70 per cent. or less of apparent typhoids. If we have a patient with no symptom but fever, but whose blood gives a Widal reaction, we say he has typhoid fever, yet we have such cases that do not react to typhoid culture and are undoubtedly cases of thermic fever. Yet when we have a case with typhoid symptoms and no Widal we say that the test has failed. In the first instance we reject the negative clinical evidence and in the second we reject the negative laboratory evidence; we do not question the reliability of the Widal test in the one, but we do in the other.

As regards malarial infection, we are by no means a unit as to the diagnostic value of a few infected red corpuscles. We certainly do have latent "malaria," though, perhaps, never without symptoms, and any intercurrent disease may drive the parasites into the peripheral circulation; we attribute the fever to the

parasites and cinchonize, sometimes with effect on the fever, sometimes without. The result is conflicting opinion as to the value of quinin in malarial fevers. The so-called malarial leucocyte count is not always characteristic; I have found it repeatedly in fevers giving the Widal reaction when there were no plasmodia. If we make a spread by drawing one slide lengthwise of another and count only within the leucocyte fringe we find relatively less small lymphocytes, because these are left behind with the red cells.

SUMMARY.

We might summarize our paper, then, as follows:

1. A very sudden hyperpyrexia in midsummer is more apt to be sunstroke than "congestion." Do not give quinin without cause.

2. The malarial leucocyte count is only collateral evidence of malarial infection, and the count must include all the cells in the spread.

3. The Widal test may lead us into error in a small percentage of cases; the blood culture test is more difficult and has no negative value.

4. A chill or fulminant onset of a typhoid fever is not sufficient evidence of a complicating malaria.

5. A chill or sudden disturbance of the temperature curve during convalescence in typhoid fever is not necessarily malarial.

6. In a malarial infection the persistence of an irregular subcontinuous fever after thorough cinchonization is not evidence of a typhoid complication or of remaining plasmodia.

7. A positive diagnosis of summer-fall fevers is not always possible.

8. In the hygienic management of summer-fall fevers we should presuppose a double infection, irrespective of symptoms.

9. Some of the summer-fall fevers are typhoid, some are malarial and a few of them are neither one, but the writer is opposed to the belief in an X fever.

A CLINICAL OBSERVATION OF NINETY CASES OF TYPHOID FEVER.

WITH FOUR DEATHS, WITH SPECIAL REFERENCE TO THERAPEUTIC FASTING.

R. M. HARBIN, M.D.
ROME, GA.

While a statistical study of typhoid fever is not in strict accordance with the caption of this article, there is a valuable amount of information as to the etiology of the disease furnished by the vital statistics of the Twelfth Census Reports and, for a practical observation, a few facts may be referred to without impropriety.

The geographical distribution of typhoid fever shows a greater prevalence in the southern states, including the Carolinas, Georgia, Alabama, Mississippi and Tennessee, the death rate being 76.1 per 1,000 deaths. Next highest mortality is 69.5 for Louisiana, Missouri, Arkansas, Indian Territory and Texas. Of the latter 80.6 is rural and 38.7 is cities. The general rate for rural is 48 and cities 19, which suggests a greater source of infection in the rural districts. Nearly every source of the infection can be excluded except the drinking water, which is contaminated by the soil. My own observation of the cases herein reported warrants that belief, as 79 cases drank well water and 11 cases drank hydrant water, and the greater proportion of the population in the locality where this observation was made is dependent on hydrant water for drinking purposes.

It will be observed in Table 1 that the general death rate per 1,000 deaths from typhoid fever is 34. In

Georgia the rate is 58, while New York has 13 and Massachusetts has a rate of 12. Atlanta, Ga., has a rate of 40, while New York City has 8.

TABLE 1.

	Population.	Deaths, All Causes.	Typhoid Deaths.	Tuberculosis Deaths.	Typhoid Deaths per 1000 Deaths.	Tuberculosis per 1000 Deaths.	Typhoid Deaths per 100,000 Pop.	Tuberculosis per 100,000 Pop.	Death Rate.
United States..	75,994,575	1,039,094	35,379	109.750	34	106	33	190	. .
White.	66,809,188	892,092	28,848	87,673	32	98	43	131	13
Colored.... . .	9,185,387	147,002	6,531	22,077	44	150	71	251	16
Georgia.... . .	2,226,331	26,941	1,535	2,651	58	99	71	120	12.4
White.. . .	1,181,294	13,094	942	981	71	74	79	83	11
Colored... . .	1,035,037	13,847	643	1,671	46	126	62	161	13
Atlanta.... . .	89,872	2,387	98	309	40	125	108	342	26 6
White.. . . .	54,090	1,250	53	119	42	95	97	220	23
Colored.... . .	35,782	1,137	45	181	39	159	125	505	31
New York State	7,268,894	130,268	1,776	14,306	13	109	24	196	. .
New York City.	3,437,202	70,229	594	8,482	8	120	17	246	. .
Massachusetts.	2,805,346	49,756	625	5,370	12	107	22	120	. .

As will be observed, the death rate in Georgia is less for the colored race, being 46, while that of the white is 71. The disease is evidently less frequent and less fatal in the colored race. This disease, excepting tuberculosis and pneumonia, claims more victims than any other disease in the south and, excluding the infant mortality of pneumonia, it is second in causing more deaths in adult life. According to death rates given by the census report in the rural districts of Georgia, there were 1,325 deaths from typhoid fever, while tuberculosis claimed 1,668.

A greater number of deaths from typhoid fever occur in the months of August, September and October and there is a more frequent occurrence after a season of heavy rainfall. It is pre-eminently a disease of early adult life, and the average age at death of all cases in the United States is 28.8 years. Typhoid fever shows a marked predilection for occupations. As will be observed in Table 2, farmers have the highest death rate, 58 per 1,000 deaths, excepting teachers, which is 98. Nurses have a rate of 56, miners 43, clergymen 37, and

physicians and lawyers 23. It will be observed that physicians, although in contact with the disease, do not often become infected and nurses are not more often infected than other classes. There are more people ill with typhoid fever during the year than any other disease, especially farmers. Estimating the mortality of typhoid at 15 per cent., tuberculosis 75 per cent. and pneumonia 20 per cent., there were farmers ill from typhoid fever 37,710, from tuberculosis 14,735, and from pneumonia 55,003. The same observation is noted for other occupations.

The death rate per 100,000 population for various

TABLE 2.

	Deaths, All Causes.	Typhoid Deaths, per 1000 Deaths.	Tuberculosis Deaths, per 1000 Deaths.	Pneumonia Deaths, per 1000 Deaths.	Typhoid Cases, 15 per cent.	Tuberculosis Cases, 75 per ct.	Pneumonia Cases, 20 per cent.	Registration Area.		
								Typhoid Deaths per 100,000.	Tuberculosis Deaths per 100,000.	Pneumonia Deaths per 100,000.
Farmers. .	96,502	58	106	114	37,710	14,735	55,003	32	147	140
Miners....	5,895	43	97	127	1,709	820	30,000	38	120	77
Physicians	2,322	23	93	87	347	306	1,011	33	168	178
Clergymen	2,062	37	98	95	503	291	980	46	123	255
Lawyers .	1,508	23	107	72	233	213	541	31	139	132
Teachers .	1,256	98	208	95	820	381	600	. . .	182	144
Nurses . .	582	56	103	91	220	90	212	47	100	93

occupations is as follows: Farmers, 32; miners, 38; physicians, 33; clergymen, 46; lawyers, 31, and nurses, 47.

These facts may be summarized as follows:

The death rate from typhoid fever in the south is startling and claims about an equal number of deaths among the whites as tuberculosis, and well water is probably the source of infection.

There are more adults ill from typhoid fever during the year than any other disease in Georgia.

Farmers, next to teachers, have the highest death rate.

The average age at death is 28.8 years.

Most writers agree that typhoid fever is a water borne

disease. The *New York Medical Record,* in an editorial, says: "While agreeing with a majority of investigators, we believe that water is the chief agent in the causation and dissemination of typhoid fever, and we admit that it may be air borne, being transmited by dust, flies," etc. Dr. Leigh Canney in a recent book regards the question of etiology as a question of pure or impure drinking water. Flies among the poor furnish a means of conveyance of infection, as the writer has often observed cases imported in families in small houses, and the disease spreads to other members of the family.

In this report of cases only clinical diagnoses were made and the suggestion of Professor Osler was adopted, viz., any fever continuing beyond ten days and resisting the influence of quinin may be assumed to be typhoid.

I regret not having a Widal test of these cases to report, but, after all, the clinical diagnoses may be quite as reliable, as only 70 per cent. give a Widal reaction before the end of the first week, according to the New York health department. All the discussions and controversies prove that no routine drug treatment of typhoid fever has any specific value. Of course symptoms should be controlled by appropriate remedies.

Turpentine was the only routine drug used in these cases, and it certainly relieves the intestinal symptoms better than any remedy at our disposal. It has been used longer and more widely than any other remedy, and its popularity still maintains with clinical observers of long experience.

Dr. Baird, at a meeting of the Georgia Medical Association, 1902, gave a résumé of the treatment of typhoid fever by 24 authorities and may be summarized in a few words: rest, diet, cold bath, etc. My own experience convinces me that the above summary represents the facts of the question, and it behooves us to study more closely the details of the rest, diet and bath treatment and our results would give us a greater percentage of recoveries.

As seems to the writer the dietetic treatment is the most important of all and no drug can counterbalance the baneful effect of dietetic sins. Fasting as a therapeutic measure has proved so valuable to me in the treatment of these cases that I account for the low rate of mortality by no other part of the treatment. The pathologic anatomy of typhoid fever shows a condition of gastrointestinal catarrh with hyperplasia, with a great reduction of all the digestive fluids. Saliva is diminished and hydrochloric acid is usually absent in the gastric juice and, in fact, almost every condition unfavorable to digestion exists. Diarrhea is an effort of Nature to throw off what the intestines can not digest. Intestinal ulcers should be assumed to exist in every case, however mild, and the proper treatment of ulcers is rest.

The rational treatment of such a condition is a restricted diet or fasting, then peristalsis can be better controlled by cold application and an opiate may be used if much diarrhea exists.

The eliminative treatment, so called, is irrational and would be unnecessary if there was no surplus of food in the intestines to dispose of.

The active symptoms in the first stage of the disease can be better controlled by fasting for 24 to 48 hours or even longer, than by any other remedy and the effect of any other remedies applied will be greatly enhanced. The effect of the cold bath is much more marked. I have often noticed that the temperature can be reduced 2.5 degrees by a simple sponge bath. Many patients exhibit marked peculiarities as to the effects of cold applications, most cases having an agreeable effect during a high temperature, while in others having an exciting effect. The Brand system being more or less impracticable, recourse is had to the modified cold bath or even a hot bath. Each case should be studied on its own merits and the cold, warm or hot sponging applied accordingly. In fact, some observers have claimed better results from the hot bath. The patient's comfort should

not be lost sight of in the bath treatment. It has been my observation that cold is better for some, while warm sponging is better for others, and still in other cases cold and hot sponging alternating has been better. In sthenic cases, with very high temperature causing delirium, ice-cold applications are preferable and in later stages, where the temperature is not excessive but delirium exists, warm sponging has a more soothing effect. In asthenic cases, with hyperpyrexia, the cold and warm alternating seem to act better. Rubbing enhances the effect of these applications and heat is thereby dissipated. It has been stated that 30 per cent. of the entire quantity of blood in circulation remains in the cutaneous system and the intimate relations between the physiologic function of the skin and temperature variations of the body both throw light on the importance of sponging in typhoid fever. An excess of food in the intestines increases peristalsis and favors the absorption of toxins. The writer has observed an increase of several degrees of temperature in a quiescent case by taking a dose of castor oil.

It is a clinical fact that emaciation is less from typhoid fever than any other acute disease, and the danger from inanition is slight. A recent writer[1] observes "that emaciation seemed to be independent of the amount of food taken into the stomach," and the same observation has often been made by myself, which would show that nutrition is not dependent on the amount of food taken into the stomach. Many cases run an abortive course after the amphibolic stage under a treatment of fasting and a restricted diet. After fasting 24 to 48 hours a very restricted diet should be given, consisting of broths of beef, chicken, mutton, oysters, etc., coffee, cocoa, egg albumin, peptonoids, etc., in prescribed amounts.

Gelatin is a valuable adjuvant in the dietary of a typhoid patient. It serves three purposes: adds to the

1. Lewis: Med. Rec., vol. lxii, No. 5.

relish of the various liquids, lessens the nitrogenous waste from the system and prevents hemorrhage. A teaspoonful three or four times a day is the amount usually ordered and should be dissolved in a little hot water and then added to any liquid nourishment, meat broths, cocoa, coffee, tea, etc. It adds a richness of taste to these slops, which otherwise become tiresome to the patient. The food value of gelatin is recognized and, being in a concentrated form, serves an excellent purpose. When used, emaciation is less rapid. The hemostatic effect of gelatin is universally conceded, but to wait until an intestinal hemorrhage has occurred but little immediate good can be hoped for by its use. It should be used as a routine in all cases to prevent hemorrhage. Very aggravated diarrheic cases do not tolerate it so well, as it may increase tympany. But in these cases it may be used on alternate days in small quantities. Since beginning its use no hemorrhage has occurred. The case should be closely studied, and any excess of food will be indicated by a rise of temperature. To nourish the patient by a minimum amount of food requires a very close study. Milk should be absolutely forbidden, because no one can predict when it will form a curd, which is worse than any solid food. One of my patients had a hemorrhage in convalescence from taking milk and lime water, equal parts. I examined curds the size of a hazelnut in the bloody dejecta. The case was a mild one. It is valuable, however, diluted with two to five parts of coffee, cocoa, broths, hot water, as under these circumstances the danger of curd formation is light.

This plan of dietetic treatment should be continued until active symptoms are controlled and, as improvement advances, nourishment may be more liberally given. Solid food is a danger that offers few advantages to the patient. Nearly all recrudescences are due to dietetic errors. Fasting in late stages and convalescent stages will cause fever, as inanition often causes fever.

Below is a summary of the table of reported cases: Of the cases 87 were white and 3 colored. The youngest was 2, and occurred with 4 others in the same family, and the oldest was 60. Cases under 10 were 20; between 10 and 20, 29 cases; between 20 and 30, 20 cases; over 30, 21 cases. The average age was 19.4. There were 48 males and 42 females. As to the months of occurrence, August claimed 27; September, 21; July, 16; October, 15; November, 5; June, 4, and April, 2. Those classed mild were 58 and those severe 32. Gastro-intestinal symptoms were marked in 36 and absent in 54. Nervous symptoms were marked in 18 and absent in 72. The average duration was 19 days, and the same without relapes was 17 days. Five, or 5 5/10 per cent., of the cases relapsed, and all recovered. The relapses were ascribed to taking too early of solid food. Five cases were complicated. Three, or 3.3 per cent., had hemorrhage, two of which were slight and the third immediately fatal from getting up in delirium.

One case (No. 88) was diagnosed appendicitis and on the fourth day an apparently normal appendix was removed and the case pursued a typical course of the disease to recovery.

Case 72 had hematuria and recovered.

Case 24 had double parotid suppuration and osteitis of the humeri, and after forty-five days recovered.

Case 14 had an attack two months previous.

Of the four deaths (4.4 per cent.) Case 46, a negro, can, with propriety, be ruled out, as he was dismissed and I was called to see him in a relapse, in a dying condition, having had urinary retention twenty-four hours.

Case 57 would probably have recovered, but, being without a nurse, she got up in delirium and a fatal hemorrhage resulted.

Case 70 contracted pneumonia on the tenth day of convalescence and died a week later. It was the opinion of my colleague that there was no reinfection of typhoid fever.

Case 64, a negro, alcoholic, died on the twelfth day from asthenia.

Of 87 whites treated there occurred 2 deaths, or 2.2 per cent.

The conclusions to be adduced are as follows:

1. Fasting and a restricted diet are indicated because of pathologic conditions.

2. A clinical fact maintains that emaciation occurs independently of the amount of food taken.

3. All severe cases should be subjected to fasting for 24 to 48 hours to relieve the active symptoms, which exhaust the patient more rapidly than the lack of food.

4. After a fast should be prescribed a restricted diet of broths, diluted milk, etc., in definite quantities.

5. Gelatin prevents too rapid emaciation in certain cases and renders hemorrhage less liable.

6. The cold bath or the modified cold bath is more effective during a fast.

7. Peristalsis favors the absorption of toxins and cathartics should be used only to remove undigested food.

8. The presence of intestinal ulcers should be assumed to exist in every case and the proper treatment is rest, which is better attained by fasting and a restricted diet, thus preventing hemorrhage and perforation.

9. The presence of diarrhea and vomiting indicates the adoption of the fasting treatment.

10. Fasting and a restricted diet shorten the course of the disease and many cases run an abortive course after the amphibolic period.

11. Many of the vaunted cures from specific drugs are dietetic in fact.

12. Recrudescences are nearly always due to dietetic errors.

13. In the above report, 45 consecutive cases occurred without death. Of 87 whites, 2, or 2.2 per cent., died. Of colored, 2 died, and the low mortality of 4.4 per cent. of all cases was ascribed to the above treatment.

TYPHOID FEVER IN COLORADO.

A REPORT OF FIVE HUNDRED AND TWENTY-NINE CASES.

J. N. HALL, M.D., AND C. E. COOPER, M.D.
DENVER.

We report to-day our conclusions regarding a series of cases of typhoid fever at an altitude of about 5,000 feet, in order to compare the disease as we see it with typhoid fever in general.

There occurred 225 cases in the private practice of Dr. Hall in Colorado, 54 of these being seen in consultation; 292 cases, most thoroughly recorded and analyzed by Dr. Claude E. Cooper, Denver, late house physician at the Arapahoe County Hospital, occurred in the wards of that institution between 1896 and 1902. We include only 12 of the cases seen by Dr. Hall in his service at St. Anthony's Hospital, the records of cases at this institution for several years being too incomplete for inclusion. He did not personally see all of the cases reported from the Arapahoe County Hospital, and he here tenders his thanks to his colleagues for permission to use the hospital records.

The males and females were almost equally distributed in the private cases, the numbers being 116 and 109 respectively. In the hospital cases males predominated, 241 to 63.

The total deaths were 77, a percentage of 14.5 per cent. The male rate was 14 per cent., the female, 15.6 per cent. That for private practice, 14.2 per cent., is unduly high, owing to the fact that nearly a fourth of them were seen with other physicians, and were naturally

grave cases. Of the 171 cases cared for by Dr. Hall in private practice 16 died, or 9.3 per cent. It is well recognized that the mortality in family practice is generally less than in the hospital, owing to many favoring conditions.

The exact cause of death can not be given in every case, since possibly pneumonia, hemorrhage and heart disease occur in the same patient. As nearly as they can be stated, however, the causes were as follows: Intestinal hemorrhage, 15; general hemorrhage, 3; hemorrhage from face (noma), 1; abortion and uterine hemorrhage, 1; perforation without hemorrhage, 7; perforation with hemorrhage, 7; pneumonia, 14; convulsions, 2; edema of larynx, 1; nephritis, 1; organic heart disease, 2; accidental poisoning by irregular practitioner, 1; heart failure (in three cases after the subsidence of the fever), 7; general toxemia and exhaustion, 15.

Arranged by age, the distribution of the cases is as follows:

8 months of age	1	31 to 40 years	108
2 years of age	1	41 to 50 years	25
3 years of age	5	51 to 60 years	8
4 years of age	2	61 to 70 years	3
5 years of age	9	Age not given	5
6 to 10 years	20		
11 to 20 years	128	Total	529
21 to 30 years	214		

The great preponderance of cases in adolescents and young adults is very noticeable, over 85 per cent. occurring between 11 and 40 years of age. The deaths were distributed as follows:

Under 5 years	0 or	0 per cent.
Between 6 and 10	2 or	10 per cent.
Between 11 and 20	15 or	11.7 per cent.
Between 21 and 30	28 or	13 per cent.
Between 31 and 40	18 or	16.6 per cent.
Between 41 and 50	10 or	40 per cent.
Between 51 and 60	1 or	12.5 per cent.
Between 61 and 70	1 or	33.3 per cent.

In two fatal cases the age is not given. Thirteen of the cases had had a previous attack of the disease, and two have had subsequent attacks, one being reported to us as fatal twenty months after the attack recorded here.

Relapse occurred in twenty-three cases, according to the records. We are practically sure that this percent-

age, 4.3, is rather too low, for the charts are not all complete. But three of these were fatal, one patient recovering after two relapses, and a total febrile course of four months.

As to symptoms present the records are not complete, and only positive or negative statements are here given.

Rose spots are noted in 270 cases.
Enlarged spleen in 155 cases.
Distended abdomen in 168 cases.
Diarrhea in 119 cases.
Constipation in 77 cases.
Unusually high temperature in 34 cases.
Medium temperature in 196 cases.
Moderate temperature in 103 cases.
Three cases are noted as having temperature over 106, and all died.

Many of these cases were seen before the introduction of the Widal test. It is recorded as positive in fifty instances, but negative findings are not noted. No cases of paratyphoid fever were recognized.

Delirium worthy of record is noted in fifty-eight cases, of which twenty-two died. It does not appear to be an especially dangerous symptom. Distinct chills are noted in fifty-three cases, and chilly feelings in thirty-one. In four of those in whom repeated chills occurred during the course of the disease phlebitis was noted, while two cases with severe paroxysms had none. In one woman the chills were erroneously thought to be septic at first, owing to a recent miscarriage. Phlebitis is noted in twenty cases, but in some of the hospital cases the records are certainly at fault, for Dr. Hall has had as many as five cases on hand at one time in the county hospital. We think this number should certainly be doubled. It is apparently more frequent in the cases that are treated by tubbing. Phlebitis is certainly much more common here than the statistics commonly given would lead one to expect. In January, 1899, Dr. Hall had a patient in St. Luke's Hospital with this complication, and every patient admitted during the previous fall had suffered similarly, probably eight or ten cases in all. But three

patients with this complication died. Two cases with double phlebitis recovered. In one case the trouble extended to the veins of the left hip and loin.

Lobar pneumonia occurred in twenty-three cases, fourteen of these dying. No exact record was kept of other pulmonary complications. Hemoptysis occurred in five cases, one of these only being tuberculous. In one case lobar pneumonia was present, in one abscess of lung, while in two the bleeding was unexplained. Two of the cases died, the one with pneumonia and one uncomplicated case.

A distribution of rose spots so general as to resemble the eruption of measles was noted in four cases, while in many others they extended to the limbs, though not to the face.

Pregnancy complicated seven cases, while two developed during the lying-in period. One woman was delivered of a healthy child while the temperature was 104, but recovered without complications. One at the beginning of the seventh month of the pregnancy had a severe attack, with three hemorrhages and relapse, but bore a living child at full term. One in the eighth month died of chronic parenchymatous nephritis, the immediate cause being laryngeal edema. She had two hemorrhages from the bowel, and the Widal test was positive. One died of intestinal hemorrhage in the fifth month. Two cases in the fifth month passed safely through the disease, and one aborted early in the pregnancy and died. Of the two cases developing in childbed one recovered.

In eleven cases such nervous manifestations occurred as frequently give rise to the diagnosis of meningitis, and curiously ten of them recovered. In one case Dr. Hall coincided with Dr. C. G. Hickey in a probable diagnosis of tuberculous meningitis, but the woman developed a severe attack of typhoid fever and recovered. One woman screamed for many days with headache when not under the influence of morphin or other drugs, but after many tub baths made a perfect recovery.

Great interest centers about the heart in this fever. Five cases were complicated with valvular disease. A boy of 13, as he recovered from scarlet fever, came down with typhoid. He died of complicating pneumonia at about the fifteenth day. He presented the systolic thrill and harsh murmur, with cyanosis and dyspnea, of congenital pulmonic stenosis. A girl of 5 years with mitral regurgitation following rheumatism passed safely through the disease, as did a girl of 15 with aortic and mitral leakage of very moderate degree following chorea. A girl of 17, not treated by Dr. Hall, but seen once by him in an emergency and not included in these figures, died in the height of the fever of well-marked aortic regurgitation, being mentioned merely to show the danger of prolonged fever even when compensation has been perfect. A man of 23 with well-marked aortic incompetence recovered. One woman with serious mitral disease recovered from the fever, but died of cardiac failure a few weeks later. One woman of 37 with a very mild attack died of sheer cardiac weakness on the seventeenth day, with absolutely no complications. Neither Dr. Hickey nor Dr. Hall could find any possible cause for such an unusual death, excepting a previous starvation diet. The woman had gone mad over some of the pernicious literature recommending a diet of fruits, and had literally lived on such a diet for months before her death. When doing obstetric work Dr. Hall frequently saw women so weak as to require the forceps from this cause, but did not realize the danger of the writings mentioned as regards typhoid fever.

One man and two women died of cardiac weakness after the fever had long disappeared, and a woman of 67 had serious general edema from cardiac failure during her convalescence, but recovered on being sent to her home in a lower altitude. A feeble girl of 8 years died of hemorrhage from a facial artery due to noma.

Two cases with purulent otitis media recovered. One patient with pleurisy with effusion recovered without

tapping, and one died, but we have no record of the exact cause. Gangrene of the right leg from popliteal obstruction necessitated amputation in an epileptic of 36 years. Suppuration of the hip joint occurred in a boy of 15, proving fatal, and a man of 22 with suppuration of the left elbow recovered. The epileptic male just mentioned, another male of 20, and a female of 23 recovered from attacks of confusional insanity following the fever. Since these statistics were gathered a girl of 22 in the insane wards of the county hospital recovered from the mania for which she had been committed months before, as she convalesced from an attack of typhoid.

One male of 28 had acute nephritis of great severity after his relapse, but recovered. The woman already mentioned as being pregnant, and an alcoholic man of 36 died with chronic parenchymatous nephritis during the disease. Many patients presented albumin, but we have no accurate record of its occurrence.

Fourteen fatal cases of perforation have been mentioned. One of these, a boy of 17 who entered the hospital twenty-four hours after the accident, was promptly operated on by Dr. Leonard Freeman, but died, as we had reasons to expect he would do. An autopsy proved that a single ulcer existed but a few inches from the cecum. It would have been an ideal case for operation if only seen earlier. A male of 29 years also died after operation, while a man of 23 recovered after operation by Dr. S. D. Van Meter. He died of fecal obstruction less than a year later, from a band of adhesions about the former wound. A man of 26 whom Dr. Hall saw with Dr. T. E. Taylor, recovered from a typhoid perforation, an ischiorectal abscess opening just to the right of the anus, as a result of the walled-in infection. This had not closed nine months later. After careful consideration we feel sure that this was typhoidal, and not appendiceal, although no Widal test was made.

False symptoms of perforation are noted five times.

A boy of 17 had screaming pain in the right flank, rigidity and rise of temperature, but no further symptoms and recovered promptly. A man of 25 had similar pain, partial disappearance of liver dulness, a leucocytosis of 13,000, and then a subsidence of all symptoms. A man of 37 had similar symptoms in his second relapse, and recovered, and at autopsy in a similar case a local peritonitis without perforation was found. In a woman of 30, at postmortem examination, we found some blood in the bowel, though no external hemorrhage had occurred, and bubbles of gas under the peritoneal coat of the small intestine, but no actual perforation. She had presented symptoms pointing toward this accident before death, but had no leucocytosis.

The occurrence of petechial eruption in typhoid is rightly regarded as rare, and as usually of fatal import. In five of these cases this phenomenon was seen, three of them dying. A male of 26 had hemorrhages from the mucous membranes as well, and died. A boy of 16, comatose from the start, had petechiæ limited to the chest, dying in the second week. A mulatto male of 21 with many subcutaneous hemorrhages had widely dilated heart and loud mitral murmur. Dr. Hall suspected malignant endocarditis from the sudden cardiac developments, but autopsy showed only severe and typical typhoid lesions. A male of 26 with many hemorrhages under the skin and edema of the feet, and a male of 39, with a moderate fever and petechiæ, recovered. This rare complication is obviously not to be regarded as necessarily fatal.

Five patients are noted as suffering from chronic pulmonary tuberculosis. A male of 28 died with a pleural effusion, as already mentioned. A female of 42 recovered, as did three males of 19, 28, and 27 years respectively, the latter having a pulmonary hemorrhage during the fever.

Two males aged 20 and 42 years respectively passed segments of beef tapeworm, and both recovered. In a

male of 33, who recovered, the plasmodium malariæ was found. He had active chills superadded to his febrile temperature.

Several suppurative conditions are of interest. General furunculosis during recovery is noted but once, but it must have occurred oftener. Abscess of the right parotid gland, opening through the ear and afterward opened externally, occurred in a man who recovered. Noma proved fatal in a feeble girl of 8. An abscess in the left groin from bedsore resulted fatally. Two abscesses of the axilla, one of the breast, one of the neck, and one in the erector spinæ muscles near an old bullet wound, are noted, all the patients recovering. The patient who had acute nephritis had abscesses in the bursæ about the left elbow and right wrist, and one in the buttock, but recovered. The typhoid bacillus was not found, but the cocci of suppuration were abundant.

Peripheral neuritis is recorded in three cases, all recovering; once in the left arm, once in the legs and feet, and once limited to the parts supplied by the ulnar nerves. But three cases of the peculiar neuritis described as "tender toes" are noted, but we recall more than that in the county hospital. They were exclusively in patients who were tubbed, all recovering.

We shall mention briefly some of the rarer complications and curiosities of the series. One woman had a wandering left kidney, with severe Dietl's crises. She became pregnant in convalescence, and no further symptoms from the kidney came to notice. One woman with distinct appendicitis developed typhoid during convalescence. In one woman a large external pile sloughed off. One young man died while vomiting. One woman of 41 died with intestinal obstruction during a well-marked attack of typhoid. Impaction of feces in the rectum required manual removal in one woman. Erysipelas complicated one fatal case. In a boy of 16 who recovered the spleen reached to the median line, but the cause, almost certainly not typhoid, is not specified. In

a boy of 10 years jaundice with some enlargement of the liver took place, presumably of the catarrhal variety. He recovered. Pericarditis with fatal result occurred in a woman of 34. Gonorrhea in a male patient pursued its normal course. One patient with serious hiccough recovered. In two instances six cases of the fever occurred in one family. One patient, after a severe attack, had acute lobar pneumonia, affecting in succession three separate lobes, and recovered after months of illness. Marked muscular rigidity was noted in one of the fatal cases with petechiæ. We regard it as an ominous symptom. A female of 18 died in convulsions on the ninth day, and a male of 31 years. In neither was kidney disease recognized.

The treatment in general was symptomatic, the temperature being generally controlled by bathing. It was interesting to compare the death rate among those tubbed with that of those treated by sponging and by electric fan baths.

One hundred and twenty-four were treated by tubbing at a temperature of 70, or as near that as could be used whenever the two-hourly temperature rose to 102.5. Alcohol was given just previous if the heart was weakening perceptibly. This treatment was used in the severer cases generally, and was applied almost exclusively in the county hospital patients. Yet but eighteen of these patients died, a percentage of 14.5, which is exactly the average for all the cases. We regard this as a great triumph for the method, for the average death rate of all county hospital cases was 15.1 per cent., including many of the milder cases not tubbed.

The causes of death of the eighteen patients submitted to tubbing were: In three cases pneumonia alone. In eight cases, intestinal hemorrhage. One of these was convalescent from diphtheria, and had a systolic apical murmur; local peritonitis and pneumonia occurred in one case. Pyelitis complicated another case, and perforation another. Abortion in one case. Convulsions in

one case. Perforation in one case. General hemorrhage in two cases. Long-continued pyrexia in one case. Nervous complications and relapse in one case. It is obvious that hemorrhagic complications are less under control by tubbing than other causes of death, and probably perforation as well.

About 350 cases are recorded to have been sponged, but as practically all not tubbed were sponged to a greater or less extent, and as a fan bath was used in a few, we shall not attempt to analyze these cases further.

We may conclude from this analysis of a considerable number of cases covering twenty years in different parts of Colorado that typhoid fever is practically the same disease here as elsewhere. Many cases called mountain fever by some have been included in this series, for we know of no such disease. Typical examples have had hemorrhages, rose spots, Widal reaction and many of the complications of typhoid, and are certainly typhoid. The false name is passing out of use here.

Dr. Hall concluded in 1889, from an analysis of his first 108 cases, that typhoid fever was milder here than in the East. A wider experience convinces him that it does not differ materially in this nor in any other respect from the disease as reported from this and most other countries. He believes that diarrhea is somewhat less frequent with us in Colorado.

We shall certainly continue for the present tubbing in severe cases, the avoidance of medicinal antipyretics, the use of alcoholic stimulants when called for by rapid and feeble pulse, and the recommendation of operation in cases of perforation seen early. We are not yet convinced of the advisability of feeding solid food before the fifth or seventh day after the temperature reaches normal, excepting in cases with exceptionally mild attacks and with clean tongue.

DISCUSSION

ON PAPERS OF DRS. HAPPEL, KRAUSS, HARBIN AND HALL.

DR. T. B. FUTCHER, Baltimore—I am not convinced that the cases referred to were not a mild form of typhoid fever. We all know that typhoid fever may present a great variety of clinical pictures; we all know that typhoid fever does not, in all instances, present enlarged spleen, epistaxis, rose spots, etc. There was one point in particular that impressed me, and that was the absence of a frequent examination of the blood serum for the Widal reaction. I believe that before we exclude the possibility of typhoid fever in these cases it is very important that the blood serum should be examined repeatedly for the Widal test. This is the routine at the Johns Hopkins Hospital, an examination of the blood serum in practically all cases of typhoid fever being enforced, especially when the patients leave the hospital, and a certain proportion of the cases show this reaction on the day of discharge.

There is another point that should be kept in mind that possibly they may have a paratyphoid infection. It is well known that in a large number of the typhoid cases among soldiers of the South African war, a number of them did not give the positive reaction after the Widal test, but were considered to be cases of paratyphoid infection.

The occurrence of chills and sweating in this series of cases is of interest. With reference to the coal-tar products it should be kept in mind that they will produce chills and sweating of a fairly severe grade. Chills and sweating may occur at the onset of the disease, at the onset of complications, or at convalescence; it should also be borne in mind that the coal-tar products will produce the same. Until more convincing bacteriologic and pathologic evidence can be brought forward to exclude the possibility of typhoid fever being the cause of these symptoms I think we must be rather conservative in stating that these cases are not cases of mild typhoid fever.

DR. JAMES TYSON, Philadelphia—While it is far from my wish to make a diagnosis in cases of continued fevers in Mississippi, I feel that in 30 years' experience in the study of fevers the tendency has been to limit the number of continued fevers. Each winter it happens to me to have one or more cases of anomalous fever which elude a diagnosis for a longer or shorter time, and invariably at the end of a certain period the cases will turn out to be typhoid fever, or malarial fever, or some of the more unusual modifications of typhoid fever, such as paratyphoid or colon-typhoid. On the other hand, there can be no doubt that fever may be kept up by irritating causes in the intestines, such as indigestible articles of food, or ptomains.

I was much interested in what Dr. Hall said regarding

typhoid fever during pregnancy. In the wards of the University Hospital it has happened to me during the past winter to have had three cases of typhoid fever occurring in pregnant women, and their terminations were quite different from those we are led to expect from text-book descriptions of typhoid fever in the pregnant woman. One was a woman eight months pregnant, and she was delivered successfully of a living child and recovered. The remaining two were pregnant five months, and both passed through an ordinary mild attack of typhoid fever and recovered.

Dr. J. A. Witherspoon, Nashville, Tenn.—This question I have been studying for ten or twelve years. To say that this fever might be paratyphoid fever is begging the question. If it is paratyphoid fever it, then, is not typhoid fever. The germ of paratyphoid fever is very different from the germ of typhoid fever. However, there is a fever in the southern states which, clinically, is different from malaria or pure typhoid fever. I will detail a few of these differences. Several years ago I suggested for this type of fever "toxenteric fever." Why? Because I did not know the cause of this peculiar kind of fever. I do know, though, that it was due to some infection, possibly in the bowel, but whether it was due to some heterogeneous germs, or specific germs, I do not know. It starts out, as described in Dr. Happel's paper, but suddenly, not gradually; it does not specially occur in the season of the year that typhoid fever does; it occurs in all seasons; it does not occur at any particular age, but may occur anywhere from the cradle to the grave; one attack does not protect against further attacks, but makes one more liable. I have seen five different attacks occur in one individual. The bowels are rather regular and the prostation is early, sweating marked, with an irregular type of fever. I have seen a temperature at 9 a. m. of 105 F., and possibly by 10 or 11 o'clock, without the use of any coal-tar products, I have seen the temperature drop to 101 F. In two or three hours there might appear a sudden rise of temperature, then it comes down again, resembling a septic type of fever. I have yet to recognize the rose-colored spots on these patients which is present in most cases of typhoid fever.

The abdomen is not tympanitic, but is rather flat or scaphoid. These patients are constipated with possibly very hard masses, but there is no diarrhea such as we expect in typhoid fever. The tongue is not like that of typhoid, and is not tremulous. There is a lack of hebetude or other nervous phenomena, but rather there is present an irritableness; they exact something, and are rather hungry. Epistaxis is rare. There is a development of herpes labialis in connection with this fever which is rare in typhoid fever. I have yet to see an enlarged spleen. The affection may be paratyphoid fever, but certainly it is not pure typhoid fever.

Dr. Charles F. Mason, Fort Sam Houston, Texas—I should like to add a few words and add to the testimony of Dr. Futcher, who insisted on repeated examinations for the Widal test if we wish to exclude the diagnosis of typhoid fever in cases of doubtful nature. Where I have been stationed I have seen many cases such as described by the first speaker. It is our custom to examine the blood several times each week both for the Widal reaction and for the plasmodium, with the result that, at some time or other, we have always found one or the other. I wish to emphasize the absolute necessity for repeated examinations. We all know how frequently we find the Widal reaction evanescent.

Dr. J. L. Crook, Jackson, Tenn.—This same question has been freely discussed in our state society. In regard to the diagnosis of these cases, I wish to state that, in my experience, when they have been treated as typhoid fever is treated they practically all recovered. These cases seem to be atypical cases of typhoid fever. With regard to the therapeusis, for five years it has been my plan to institute drainage and antisepsis by the use of calomel and carbonate of guaiacol. In a series of 140 cases the mortality has been less than 3 per cent. When acetozone was used in 9 cases there was no mortality.

Dr. R. W. Lindsey, Little Rock, Ark.—I believe we have a fever in the south that is different from typhoid fever, and from malaria. It is a continued fever, lasting from three to five or six weeks. There are none of the symptoms of typhoid fever, except that it is a continued fever. It is not as grave as typhoid fever. The exacerbations are as liable to occur in the morning as in the evening. Tympany does not occur, and the bowels, as a general thing, are constipated. Until this year, or last fall, I have never had an opportunity of testing microscopically the blood of these cases. Three cases were examined for the malarial plasmodium, and the Widal test made for typhoid fever. These cases lasted three, four and seven weeks respectively. The blood of these cases was examined the first day I saw them, before any quinin was given, and neither the typhoid bacillus nor malarial plasmodium was found. The blood was taken at different stages of the three cases and examined, with like results.

WEAKNESS AND DILATATION OF THE HEART IN CHRONIC NUTRITIONAL DISEASE.

G. W. McCASKEY, A.M., M.D.
FORT WAYNE, IND.

I wish to invite your attention for a few minutes to some practical questions concerning certain aspects of cardiac pathology and therapeutics. These questions will be discussed with especial reference to my own experience. This experience, or at least that part of it which I have chosen as the basis of this discussion is perhaps sufficiently indicated by the title. It might be well, however, to add that the material among which cases of weakness and dilatation were found comprised about 600 cases of chronic gastritis, with about the same number of other forms of chronic diseases. These cases all occurred in private office and consultation practice. In all cases the heart was subjected to a careful examination with reference to the clinical changes of volume, either transient or permanent in character, which occur with varying states of tonicity of the cardiac muscle.

In perhaps no other department of practical medicine have greater therapeutic advances been made than in that relating to cardiac disease. If Senac could appear before this Association and ask, to-day, instead of the middle of the eighteenth century, his despairing question, "What can medicine do for dilatation of the heart?" he might receive for answer the records of innumerable relative or absolute recoveries. There are few, if any, better landmarks by which to measure the rapid strides of clinical medicine as distinguished from

clinical surgery, and demonstrate the lack of consideration and truthfulness of the unfavorable comparisons thus often made.

The great work of Senac was the first important stone in the foundation of modern cardio-pathology and therapeutics, but the structure was very slow in building. Its outlines only began to assume a distinct form half a century later, with the advent of Corvisart, the great physician of the great Napoleon, and later by the labors and discoveries of Laennec, Bouillaud, James Hope and others.

Until a comparitively recent date the recognized pathology of the heart had too exclusive a reference to its orifices, its valves and its membranes. The muscular tissues of the heart to which these appendages are subservient was, in effect, assigned a secondary rôle.

It would be interesting, if time permitted, to epitomize the survey which I have recently made of the manner in which the great writers on the heart during the nineteenth century, which almost exactly corresponds to the evolution of the subject, have dealt with the heart muscle, but time will permit only a few references. Corvisart said that the heart distended the same as does the bladder with urine, but only considered shock, suppression of menses and hemorrhagic flux among the causes of dilatation.[1] He observed the frequency of dilatation of the heart in tailors, tanners, washwomen and others continually bending forward, and attributed it to compression of the aorta. He really devotes considerable space to the discussion of the heart substance, but it is, of course, in the light of a century-old pathology.

James Hope[2] more clearly recognized the ordinary changes in the condition of the heart muscle in dilatation, and treated cardiac dilatation as a primary disease.

Walshe[3] gave very little attention to the heart muscle, but made the important observation that "there are cases in which the heart notably loses its consistence,

without there being any permanent blood disease or any obvious affection of the organ itself, except a deficiency in performance."

The work of Stokes,[4] published in 1854, was a great advance on any of its predecessors and really presented a very interesting and, at that time, advanced view of the morbid conditions of the cardiac muscle. He notes that the heart muscle is probaly liable to all the vital and organic changes found in muscle structure elsewhere.

The beginning of the last quarter of the nineteenth century witnessed the publication of Hayden's[5] ponderous volume of 1,200 pages, which, with liberal abstracts and epitomes of the work of his contemporaries and predecessors, fully reflected all the then existing knowledge regarding the heart. He was, perhaps, the first to fully recognize the very common clinical connection of dilatation with simple debility and softening of the walls of the heart from anemia and retrograde changes of the tissues without obstruction to the circulation. This volume is in itself an instructive, though somewhat out of date, library on the heart. I can not, however, deal further with this retrospective view of the pathology of the heart muscle.

Balfour[10] has well said, speaking especially of the heart, that the vigor of any muscle depends on the perfection of its metabolism. In practically all cases of chronic nutritional disease the metabolism of the entire body is notoriously at fault, and the heart muscle feels, with all other muscles, the loss of vigor incidental to its perversions. We are familiar with it in the lack of force and endurance of the voluntary muscular apparatus; it is conspicuous in the muscular atony of stomach and intestines, and we have learned to look for it and recognize it as a factor of vital importance in the functional integrity of the heart. In this connection it should be remembered that the nervous system, on which all muscular tissue is dependent for

trophic and motor influences, is itself equally dependent on metabolism, with all that this implies, for the proper performance of its functions, and that the heart in such conditions suffers from a perverted innervation as well.

One of the first results of this weakness, so far as the heart itself is concerned, is an inadequate systole. We know that the ventricles of the perfectly normal heart do not completely empty themselves. At the close of every systole there is a very small amount of residual blood remaining in the ventricles. The precise amount must depend on the vigor of the ventricular contraction, and must increase with progressive weakness of the cardiac muscle. In its milder grades this amounts only to exaggerated physiologic distension, and perhaps only occurs when a slight demand is made on the heart for extra work, such as a degree of exercise that would not perceptibly effect a normal heart. As the weakness increases the overdistension results from still slighter causes until, finally, in certain cases it becomes permanent and constitutes what is clinically known as dilatation of the heart.

The occurrence of dilatation is dependent on at least one other important factor, aside from weakness of the cardiac muscle, namely, the arterial resistance. The resulting intracardiac tension is subject to great variations in both health and disease, but, in the absence of organic disease involving the arteries, it is always relatively low in cases of weak heart. But even when relatively low there is considerable pressure, even in the passive state. In health it amounts to perhaps 10 to 12 c.m. of mercury; in debilitated states it may drop to 5 or even much less, but a positive pressure equivalent to several c.m. of mercury is absolutely essential to the functional integrity of the organs supplied and this pressure must be overcome by the contraction of the cardiac muscle. If the heart muscle is unable to force its contents through the arteries against this

pressure, an increasing amount of residual blood remains in the ventricles and dilatation results. It is perhaps only rarely that the weakness of the cardiac muscle is sufficient to lead to a continuous dilatation while the patient is in a passive state. The overdistension occurs in paroxysms coincident with a transient rise of pressure, usually the result of exercise. Exercise always raises the arterial tension, both in health and disease, and leads to transient dilatation, which the healthy muscle can overcome but the weak muscle can not, and in weakened conditions of the heart muscle these periods of overdistension are only partially recovered from, and when this occurs repeatedly the various grades of dilatation of the heart, slight or severe, ensue. It is in reality a frequently repeated overstrain of the heart. It is, in effect, precisely what occurs when the healthy heart is overstrained, only the results follow comparatively slight causes. Overstrain of the heart is altogether a relative affair. I think it is Stokes who relates a case in which a patient with an extremely weak heart was slowly improving by absolute rest in bed, not being even permitted to wash his own face. After considerable improvement, but, before being authorized to do so, he sat up in bed and washed his own face and fell over dead. This was simply fatal overstrain of a very weak heart from an apparently very trifling cause. In another case a similar result might follow walking across the room; in a third it would follow a short run to catch a street car or by hurrying up steps; while in a perfectly healthy heart the same result could be produced by sufficiently severe and sufficiently prolonged muscular exertion, as probably occurred in the classical case of the Greek soldier who delivered his message of victory and fell over dead. Hereditary tendencies, or at least an inherent inadequacy of the heart muscle, undoubtedly has much to do with the phenomena in any given case. Some hearts are primarily defective, just as are some

livers and some brains, and readily succumb to causes which would not be operative in other cases.

The pathology of the cardiac muscle in the cases under consideration is quite variable. Eichhorst[13] says that weakness of the heart muscle may cause death without any macroscopic or microscopic changes in its structure. However, this is perhaps rarely true. The nutritional disturbances incident to chronic disease lead to various alterations in the texture of muscles in the heart and elsewhere. Barrie says[12] that when dilatation occurs there is always granulo-fatty degeneration of the heart fibers and pigmentary infiltration. In such cases Gibson[11] and others speak of an indistinctness of the striations of the muscular fibers. Probably in all the severe cases there is some sort of change more or less marked in the cardiac muscle. While it is very desirable to have as full an understanding as possible concerning the pathology of each case, it can only be determined with a certain degree of probability. It has, of course, an important bearing on prognosis, but the existence of marked degenerative processes do not bar recovery. In many cases chronic myocarditis is probably the underlying morbid condition. The existence of certain types of chronic inflammation, as well as degenerative processes in chronic nutritional disease, is quite intelligible in the light of modern pathology. These various morbid processes are the result of subnutrition and toxins. The hemoglobin coefficient is usually low and there is scant material for tissue reconstruction, while, in addition and partly as a result, there is defective elimination on the part of the heart muscle itself of its own waste products, as well as of the body in general. The retention of leucomaines and other products of retrograde changes has a still further depressing influence on the function of the heart muscle and favors the development of the anatomical changes already referred to. But, in addition to this, and especially in the class of cases with which I have per-

sonally had to deal, we have a new factor in the introduction into the circulation of bacterial toxins produced in excess in the digestive tract. These toxins probably unite with the side chains of the protoplasmic molecules of the heart muscle, just as certain metallic substances are supposed to do with more or less impairment of functional power and, finally, with the production of genuine structural alterations of the character already indicated.

In the cases in which no microscopic alterations can be found in the heart muscle the tissues are softer and more easily torn, as long ago pointed out by Walshe.[8] It can scarcely be that such a physical change can occur in the heart muscle without antecedent structural alterations undiscoverable by our present means of research.

Although not strictly within the scope of this paper, I am tempted to briefly refer to syphilis, the very extensive prevalence of which, among all classes, makes it a frequent factor in the class of cases under discussion. Syphilitic involvement of the heart is probably much more common than is recognized, and is not a very infrequent cause of weak heart, leading to varying degrees of dilatation. Aside from the gummatous disease of the heart muscle itself, and involvement of the coronary artery, there is recognized by syphilographers a syphilitic myocarditis. And there is good reason to believe that, prior to the evolution of such gross changes as this, there is a period of weakness which, in consequence of proper treatment, may never pass into the stage of structural alteration. The hearts of syphilitic patients are very vulnerable, and this cause of cardiac weakness should receive wider recognition than is at present accorded it.

In chronic gastro-hepatic disease there are some reflex nervous phenomena which should be considered in addition to the factors already mentioned. The experiments of Morel and others have shown that, on stimulation of the terminal filaments of the pneumogastric or sym-

pathetic nerves in the gastric mucosa, or possibly of both, there is a reflex spasmodic contraction of the pulmonary capillaries, with a rapid rise of pressure in the pulmonary artery and a transient dilatation of the right ventricle. If reflex irritation of these nerve filaments is continuously produced by a pathologic lesion, Barrie[12] says it may cause persistent dilatation, which may increase until there is tricuspid insufficiency with extreme ectasia of the right ventricle. He adds, however, that its relative infrequency in gastro-hepatic disease points to an individual predisposition which may, of course, be hereditary or acquired through the operation of some of the causes and conditions that I have already considered.

It is unnecessary to occupy time in dwelling on the individual chronic diseases which occupy an etiologic relationship to the condition of the heart muscles under discussion. Among them may be mentioned leukemia, chlorosis, all the different types of anemia, either secondary or those other types which in our ignorance we call primary, chronic diseases of the digestive organs associated with subnutrition and relative failure of elimination, excessive toxic conditions, etc. These various causes act on the heart in at least four ways: First, a failure to supply sufficient nutrient pabulum to the heart muscles because of a weakening of the complicated mechanism concerned in its accomplishment. Second, through the chemical influence of retention products, which are toxic in excess, and the absorption of foreign substances, for the most part, from the gastro-intestinal tract. Third, through the partial failure or perversion of the important phenomenon of innervation of the heart muscle. And, fourth, the purely mechanical factor which is operative from the moment when the cardiac muscle is unable to make an effective ventricular contraction against the resistance offered in the arteries. Sometimes one or several of these causes will predominate, but I think they will be found to fully explain the clinical phenomena presented.

The symptoms of heart weakness and dilatation vary within wide limits. It is scarcely necessary to dwell on the clinical manifestations of fully developed and well-marked dilatation of the heart, with its well-known train of symptoms. It is more important to emphasize the fact that there can be, and frequently is, a recognizable grade of dilatation, with either an entire absence of symptoms or at least such as can be elicited with great difficulty. The earliest symptoms are simply those of a weakened circulation, and in the various types of chronic nutritional diseases, with their associated debility and slight dyspnea following a given amount of exercise, are simply attributed to the general disease present. It is not until the dilatation has proceeded to a degree which will produce valvular incompetency by enlargement of the orifices or separation of the insertions of the papillary muscles, or both, that the obvious clinical picture of heart disease with dominant heart symptoms is presented. This condition, however, is preceded by a long series of events, with any one of which, perhaps, its onward march could have been arrested if duly recognized and properly treated.

The importance of these slighter and frequently unrecognized grades of dilatation of the heart is not limited to the heart itself, but, in addition, frequently furnishes the key to the entire ensemble of symptoms, which, while having a complicated origin, are possibly based more largely on lowered blood tension than on any other single factor.

I have not attempted to make any statistical statement of the number of cases in which demonstrable grades of dilatation have been presented, as such a statement with reference to so vague and diverse a group of cases would appear to be valueless. I will say, however, that in a very large proportion of the cases which have been able to come to my office for examination and, perhaps, subsequent treatment, such a condition of the heart has been found. I should

think that possibly 20 per cent. of these patients presented demonstrable enlargement of the heart, varying in degree from an enlargement so slight as to be doubtful until verified by a decreased area of dulness under subsequent treatment, to cases so severe as to make dyspnea on very slight exertion one of the principal complaints in the case. The diagnosis is based principally on the area of deep dulness, which has, of course, to be quite accurately determined. This I do not consider difficult in the vast majority of cases. Of course where there is emphysema or excessive obesity, it becomes proportionately more difficult. Broadbent[9] says that it can not always be determined, and further adds that the so-called auscultatory percussion is of no value whatever. With the last statement I must entirely disagree. By the use of the ordinary methods of auscultatory percussion or, better, by the use of the phonendoscope, with either light percussion or friction, the borders of the heart, and especially the left, can be determined with great accuracy. I have verified the results of these examinations, both by autopsies and skiagraphs, and have the utmost confidence in them. By placing the phonendoscope in the intercostal spaces over the area of absolute dulness and making very light strokes or friction sounds gradually extending outward, the border of the heart is easily recognized. The sharpness of the line of demarkation where the transition of sound occurs is really very striking. I only mention this because there is a more or less widely spread opinion such as that already quoted from Broadbent, that slight changes in the volume of the heart can not be clinically determined with accuracy.

In the severer cases regurgitant murmurs will sometimes be found constantly present over either the mitral or tricuspid area or both. In many of the milder cases, however, where such a murmur is not heard, it can readily be produced, while in others it can be produced with difficulty, and the readiness with which it can

be elicited furnishes some indication of the tonicity and recuperative power of the heart muscle. The character of the heart sounds soon becomes modified as the case progresses, the first sound losing its characteristic qualities, more and more approximating to the character of the second, and, at the same time, the interval between the second sound and the first becomes gradually shorter, approximating more and more to the interval between the first and the second, until it finally becomes practically the same.

The treatment of these cases should have in view two principal objects: first, to improve the general nutrition and correct the defects and perversions of metabolism, which are the underlying causes, which includes rational and adequate treatment of any organ or set of organs which may be at fault, among which the digestive organs stand pre-eminent; and, second, when necessary, the application of special therapeutic measures directed toward the heart.

It would not fall within the scope of this paper to make any attempt at a discussion of the multiform methods included under the first heading. It need only be said that it includes and requires adequate nutrition, which means an ample food supply properly digested and assimilated to the fullest extent possible under the conditions presented; effective eliminationn of the products of retrograde changes which may be unusually toxic owing to morbid perversions in the chemical processes occurring in the tissues; tonic and reconstructive medication, such as the exhibition of iron and bitter tonics where these appear to be indicated; and, finally, the best mental and physical hygiene available under the circumstances of the case.

So far as the second object of treatment is concerned, it will frequently happen that no special measures directed toward the heart will be required, especially in the milder cases. Proper rest for the nervous system and the circulation conjoined with the measures already

indicated may be sufficient to restore the heart to its normal condition and size. In many cases, however, I have found such special methods of treatment not only advantageous but necessary. Where the heart is quite weak, with frequent pulse and the blood pressure low, the cautious administration of digitalis for a brief period of time has been of the greatest service to me in these cases. Other heart tonics may also be employed. Such outdoor exercise as is indicated in the case should be graduated, with special reference to the heart, and in most cases forms an indispensable part of rational treatment.

I wish now to speak of the saline carbonated baths as a means held in reserve for the severer cases or even some of the milder ones which for some reason prove intractable. I am fully convinced of their very great value after having treated a considerable number of cases in this manner. I have found, in agreement with Broadbent and others, a remarkable decrease in the area of deep cardiac dulness as a result of a single bath properly administered. In fact, I am not aware of any other possible means of influencing so immediately and directly the dimensions and tonicity of the heart. I have found the left border of the heart more than one centimeter further to the right immediately after such a bath than it was before, and have verified the findings with the fluoroscope. At the same time the pulse will drop in frequency anywhere from 15 to 30 beats, with a corresponding improvement in volume and tone. The greater part of this phenomenal change is comparatively transient, but where treatments are given daily the heart does not lose quite all it has gained, and after three or four weeks of such treatment may attain nearly or entirely its normal size and remain permanently so under favorable conditions, which means, of course, a removal of the causal factors and a continued maintenance of the general health. Gibson[11] says that it is just in this class of cases that the very best results are obtained

from the Nauheim methods. I believe this is true, and have certainly had most gratifying results in properly selected cases, although it is not used, as a matter of course, as a routine measure.

With the resistance movement I have had less experience, as my patients have, for the most part, been able to resort to active instead of passive movements; and properly graduated gymnastics and outdoor exercise have appeared to me to be all that was necessary.

In conclusion, I wish to emphasize the favorable prognosis of dilatation of the heart in most cases, even the coexistence of valvular disease not necessarily constituting an insurmountable bar; its relative frequency in chronic nutritional disease, and the strong probability that the milder cases, if unrecognized and neglected, finally present all the clinical features of advanced and incurable heart disease.

BIBLIOGRAPHY.

1. Corvisant, J. N.: A Treatise on the Diseases and Organic Lesions of the Heart, 1813, p. 139.
2. Hope, J.: Diseases of the Heart and Great Vessels, 1839.
3. Walshe, W. H.: A Practical Treatise on Diseases of the Lungs and Heart, 1851, p. 434.
4. Stokes, William: Diseases of the Heart and Aorta, 1854.
5. Hayden: Diseases of the Heart and Aorta, 1875.
6. Fothergill: The Heart and Its Diseases, 1879.
7. Clark, Alonzo: Diseases of the Heart, 1884.
8. See, G.: De Traite des Maladies du Coeur, 1893.
9. Broadbent, W. H.: Heart Disease, 1897, p. 21.
10. Balfour, G. W.: Clinical Lectures on Diseases of the Heart and Aorta, 1898.
11. Gibson, G. A.: Diseases of of the Heart and Aorta, 1898.
12. Barrie, Ernest: Traite des Maladies du Coeur, 1900, p. 629.
13. Eichhorst, H.: Practice of Medicine, 1901, p. 22.

407 West Main Street.

LIMITATIONS OF NON-SURGICAL TREATMENT IN ACUTE INTESTINAL OBSTRUCTION.

JOSEPH RILUS EASTMAN, M.D.
INDIANAPOLIS.

The term "acute obstruction of the bowel," broadly used, embraces all of those conditions, however varying in their etiology and pathology, which suddenly obstruct the lumen of the intestine to the extent of interfering with the passage of its contents. It is well known that the intestine may be obstructed by omental bands, fibrous bands, the result of peritoneal adhesions, as in appendicitis or peritonitis (*ante et post operationem*), by Meckel's and other diverticula, or at the neck of an internal or external hernia; that it may become kinked or twisted as in volvulus; that one segment of the intestine may become telescoped into a neighboring segment, as in intussusception; that the lumen of the intestine may be obstructed by gallstones, enteroliths, foreign bodies or its own normal contents; that it may be narrowed by pressure of tumors from without; that its lumen may be narrowed by benign or malignant strictures, or by the pressure of neoplasms or acute swellings arising within its own walls, and by the presence of embolism or thrombosis of the arterial or venous circulation of the bowel wall. Symptoms of acute obstruction may also develop suddenly in cases of malignant, syphilitic or gonorrheal stricture.

In all of the above enumerated conditions the obstruction may be said to be purely mechanical. Cases of ap-

parently purely functional closure of the intestine—intestinal spasm—are recorded. Heidenhain and others report a few cases of purely spastic or reflex tonic contracture. In Heidenhain's cases, the contracture seemed to be complete and irreducible. Quintard[1] collected fourteen such cases from the literature. Quintard's list is doubtless incomplete, but nevertheless cases in which complete contracture of the intestine is thus brought about by reflex irritation must be quite rare. Perhaps operation and very careful direct ocular examination in these cases would have left fewer of them recorded as of purely reflex origin. It is well known, however, that the syndrome known as ileus, as a purely reflex phenomenon, may attend or be consequent upon torsion of the pedicle of an ovarian tumor, or pinching of the spermatic cord, and that in an inorganic or functional form it may be due to ptomain poisoning or to dysentery or other inflammatory conditions in the large or small intestine. No one knows how many cases of reflex or functional interference with the procession of the intestinal contents have been included in the list of cases of mechanical intestinal obstruction cured by internal therapy.

If a surgeon, speaking from the standpoint of a limited personal experience and observation, may assume to enumerate the forms of acute obstruction of the bowel which may be cured by internal medication, the writer would include the following: First, simple coprostasis. It should be borne in mind, however, that behind the picture of simple impaction may be hidden deep strictures benign and cancerous, or compression of the colon from without by adhesion bands, etc.; second, cases of incomplete stenosis of the small intestine from chronic peritonitis. Heidenhain states that many of these cases are cured by thorough and repeated rinsing of the stomach, the use of enemata and proper dieting; third, volvulus of less than 180 dgrees when involving the colon; fourth, reflex closure of the intestinal lumen.

Medical treatment of reflex ileus is not only valid, but it is our best resource. The preparations of hyoscyamus are useful agents in this form of obstruction. A single hypodermic injection of morphin may, by one brilliant stroke, efface every symptom of a violent reflex ileus, as noted by Dr. Hugo O. Pantzer of Indianapolis. It is in ileus of the reflex type alone, however, that morphin is useful, and even in cases strongly suspected of being of reflex origin, the administration of morphin or antispasmodics should not be continued too long; fifth, functional obstruction due to dysentery or other ulcerative and irritative processes, such as ptomain or lead poisoning. In such cases the cause of the ileus may usually be removed by non-surgical methods; sixth, intussusception of the colon during the early stages.

Körte has called attention to the fact that the symptoms of incarceration of gallstones are often so similar to those of a suddenly developing ileus with obstruction of the bowel, that a mistaken diagnosis of stricture or volvulus may have been made. Celiotomy is clearly indicated in such cases. Cholelithiasis is, as is well known, a surgical disease, notwithstanding it is not necessarily fatal, and is occasionally amenable to medical treatment. The ileus produced by it should be relieved by surgical removal of the cause.

No doubt in very rare cases invagination has been reduced by the forced introduction of air into the rectum, or by high colonic injections of water, but that the vast majority of these cases must be given over to the surgeon, is, or should be, well known. A volvulus of 180 degrees in the first stage of the disease may be reduced by air or water; however, in cases of volvulus reaching 250 or 300 degrees, it becomes impossible to inject water into the closed-off segment. The stenosis is complete No one would presume to be able accurately to estimate the amount of twisting of the bowel in any given case before operation, or to specifically diagnosticate volvulus at all. As for internal stenosis due to organic strict-

ure, it is hardly imaginable that such a case could be successfully treated by other than surgical means, and it is generally taken to be undebatably concluded that obstruction due to bands, diverticula, strangulation of herniæ and the presence of neoplasms must be dealt with surgically.

It would be obviously very difficult accurately to determine the number and character of cases spontaneously relieved or cured by non-surgical means, since after the subsidence of symptoms and cure of the patient, the abdomen not having been opened, the nature and cause of the trouble must remain in the dark. We find ourselves here on the ground of pure hypothesis. We know how difficult it is to determine the exact nature of an obstructing intestinal lesion by consideration of the external signs and subjective symptoms alone.

In cases of definitely diagnosticated mechanical obstruction of the intestine of any sort, there are few surgeons who, with instruments at hand, would adopt any less radical measure than that of operation. The non-operative methods of treatment may, under very extraordinary circumstances, be used in mechanical obstruction by one who, for various reasons, feels that he dare not open the abdomen. For one, however, with proper surgical means at his disposal, to stand by with folded hands, or deal with these cases by the so-called palliative means, requires upon his part a reckless degree of indifference indeed. The palliative means are nearly always ineffectual in cases of mechanical obstruction, are often deceptive in their effects, and are never free from danger.

Even very young children stand abdominal operations remarkably well, and there is no reason why a celiotomy, if indicated, should be withheld from an infant, even if it be only a few days old. Here the methods of inflation and injection are not devoid of danger. Knaggs reports eight instances of fatal injection. In seven of these ruptures occurred. In one case, that of a young infant, only

nine ounces of water were injected when a rumbling noise was heard in the abdomen, which was immediately followed by death. (Maylard.)

It will appear to any one familiar with the anatomico-pathologic nature of the condition that the reduction of intussusception of the small intestine by the injection of water or air will hardly be possible after the onset of symptoms. It is doubtful whether in the adult it is possible to force water through the ileocecal valve. In the cadaver this may be done. Here the valve may leak, but it should be remembered that this phenomenon, though it may be demonstrated by the use of force in the cadaver, can hardly be produced in the live subject.

It must be conceded that in acute intussusception, the lesion being almost always in the small intestine, little can be gained by the injection of water. Irritating enemata may stimulate peristalsis in the lower intetine, and the movements may in some degree be transmitted to the small bowel. This will hardly suffice to release the intussuscepted small intestine if sufficient time has elapsed for the establishment of a clinical diagnosis. For by this time the intussuscepted bowel will have become quite thickened and choked, or even gangrenous. The employment of massage or taxis blindly through the abdominal coverings has never accomplished anything in any case of intussusception of which the writer has any personal knowledge. The toughness of the adhesions between the layers of the intussusceptum and the sheath or intussuscipiens and the rapidity of their development are extreme.

A few cases in which symptoms of intussusception had existed for from six to nine days have been, according to reports, relieved by the combined conservative methods. The Almighty only knows how many thousands have been lost through the employment of these so-called, but miscalled, conservative methods. Operation will conserve the lives of a certain small percentage of cases of intussusception. It represents, therefore, in the

strictest sense, conservative treament. Many cases of successful excision of acute intussusceptions have been reported, and in many more cases lives have been saved by the employment of the at once less brilliant and less dangerous methods of operation.

A favorable result sometimes follows irrigation of the stomach and colonic injections of water, or insufflations of air. The physician permits himself to be deceived by the comparatively good condition of the patient, and operation is postponed until the patient is moribund, then an operation is allowed and surgery is charged with a mortality.

Successful treatment of intestinal intussusception is only possible by operation performed early, while tympanites is still slight and the patient is in relatively good general condition. Later, distension of the intestine becomes so great that often, with the abdomen opened, the most careful and zealous surgeon can not find the obstruction at all.

It would be well if there were unanimity of opinion among surgeons and physicians as to the proper treatment of a malady in which the mortality is not less than 75 per cent.

Laparotomy in such a case is always a dangerous operation, but the disease unoperated is distinctively more dangerous.

Nature has shown us the way to deal with intussusception. When, as rarely happens, she effects a cure, she does it by sloughing and gangrene and discharge of the intussuscepted segment through the bowel. It may seem quite superfluous for one to declare at this day the necessity for prompt diagnosis and prompt operation in cases of intestinal obstruction of this character. But the fact is that physicians, as a rule, are not promptly making differential diagnoses in these cases, nor operating them until the patients are *in extremis* with subnormal temperature, flickering pulse and abdomens blown out as tightly as a snare drum.

Already many successful operations for intussusception in young children are recorded. Karrsberg[2] operated on a boy 3 years and 9 months old, with invaginatio ileocecalis subchronica with complete success. Bryant[3] describes a case of acute double intussusception in a girl baby of 9 months, operated on twenty-four hours after the onset, with rapid recovery. A. B. Mitchell met a case of retrograde intussusception in a boy 4 months old. It was reduced by operation thirty-six hours after the appearance of symptoms and recovery followed. Wright and Renshaw[4] reported successful operation on a 3-year-old child with invagination of the appendix into the cecum, and Eve successfully operated on an intussusception at the ileocecum in a baby. Poppert of Giessen saved an 8-months-old child with intussusception by prompt operation, and Sells[5] also operated successfully on an infant 4 months old, 18 inches of the ileum having passed through the ileocecal valve. Several dozen similar cases have been reported. Northrup, on the other hand, reports relief of a child seven months old with intussusception by high colonic injections of water, and Ouvry[6] successfully treated a case of acute colonic invagination in a male infant 9½ months old by catheterization of the invaginated bowel.

The surgeon, however, will ask, with a shrug, whether these and other similarly palliatively treated cases were really intussusceptions. Relief in Northrup's case followed free movement of the bowels and the only indications of intussusception were a sausage-shaped tumor under the liver and bloody dejections.

Fitz's statistics give fifty-one cases treated without operation, with sixteen recoveries and thirty-five deaths (69 per cent.). Pilz reported in 1870 ninety-four cases

---

2. Hospitalstidende, 1896, No. 5.
3. Lancet, Oct. 12, 1901.
4. British Medical Journal, June 12, 1897.
5. Lancet, Aug. 31, 1901.
6. Gaz. Hebd., June 20, 1901.

in patients under 1 year, with a mortality of 84 per cent. From 1870 to 1891 135 cases in patients under 1 year gave a mortality of 59 per cent.[7] More recent statistics would show a much smaller percentage of mortality after operating, as the result of earlier and more skillful surgical intervention.

A case of invagination reported by Jaboulay[8] is instructive, for the reason that no operation was made, and the autopsy showed that surgery would doubtless have saved the patient's life. The case was received into the clinic forty-eight hours after the appearance of symptoms. There was no stercoraceous vomiting. On the following day there were bloody stools. Operation was withheld because it was assumed that after seventy-two hours the vitality of the intestine must be practically lost, and, moreover, the lesion could not be accurately located. Administration of opium was followed by improvement, then the symptoms grew steadily worse. At the autopsy on the tenth day there was found a high intussusception (about 18 inches from the stomach) which was readily released by delicate traction. The intestine was in relatively good condition.

When considering operation, it often becomes a difficult matter to determine whether a given condition of ileus has been mechanically produced, or produced by peritonitis, for it is well known that the most vicious peritonitis may proceed without fever. Lejars has called attention to the fact that tubercular peritonitis may produce all the phenomena of intestinal obstruction without the slightest mechanical obstruction being present. Here the doubt should weigh in favor of operation, for since the operative treatment of peritonitis promises relatively good results, the operation is therefore specially indicated. In point it may be mentioned that Turner[9] operated on a case of obstruction due to

7. Fischer, N. Y. Med. Jour., Feb. 21, 1903.
8. Province Med., 1896, No. 5.
9. Lancet, April 12, 1902.

tubercular peritonitis with prompt recovery and rapid disappearance of the peritoneal tubercles.

Volvulus or twisting of the intestine is a comparatively rare cause of obstruction. There are, in most cases, no symptoms sufficiently distinctive to admit of differentiation from obstruction due to other causes. Were it possible to diagnosticate these cases, they would in the very nature of their development and formation lend themselves rather favorably to treatment by abdominal taxis, or if in the colon, to treatment by injection or inflation. Rigorous shaking with manipulation of the abdomen, and stimulation of peristalsis by enemata may rarely untwist the involved gut. As a matter of record, however, it is rarely indeed that anything avails in severe cases except operation, and the same rules regarding early operation should apply here as apply in cases of mechanical obstruction of other forms.

The direct untwisting of the volvulus before great thickening and destructive changes have taken place in the obstructed part is often comparatively easy. A few days will place its reduction beyond the highest skill of the surgeon.

In early operation lies our greatest hope in all cases of mechanical intestinal obstruction. Naunyn collected from the literature 288 cases of operation for ileus. From the record of these cases it appears that by far the best results were obtained after operations on the first or second day after the beginning of symptoms. Results of laparotomy for intestinal obstruction in individuals suffering from hernia were particularly fortunate. These were, of course, not cases of incarcerated hernia, but cases of ileus as the result of stasis, adhesions and torsions in the neighborhood of the hernial openings.

The reason for the surprisingly good results in these hernia cases is to be sought in the fact that the operators knew where to look for the obstruction, and operated early. This fact forcibly impresses one with the value of

accurate localization of the obstruction before operation, and early operating. The fact seems to stand forth in very clear light that in all diagnosticated cases of mechanical obstruction, and all cases in which there is doubt as to the nature of the obstruction, the so-called conservative methods, the administration of morphin, stomach lavage, injection and inflation of the colon are not only almost valueless, but often really dangerous. Morphin is especially treacherous. In giving it we "fall back upon the soft pillows of Providence," in most instances stultified and deceived, awaiting the messenger with the inverted torch.

In paralytic reflex ileus following abdominal operations, it must be conceded that the expectant plan of treatment is the better to follow. Unfortunately, it is not easy to distinguish this form from mechanical ileus; moreover, the former may develop into the latter. Here, again, doubt must weigh in favor of operation.

Post-operative adhesions producing obstructions must be dealt with promptly by reopening of the abdomen. In such adhesions are assumed to be present when oil and salts in generous doses fail to pass the intestine, and high colonic injections administered by one who really understands how to fill the colon with water fail to give relief.

The value of laparotomy as a purely diagnostic measure should not be underestimated. Notwithstanding Treves, Maylard and others have laid down very useful rules and precepts for differential diagnosis in intestinal obstruction, it is nevertheless very well known by those who frequently deal with these cases that the exact nature of the lesion is rarely made out before anesthesia has to some degree relaxed the abdominal wall, and usually not until the abdomen has been opened and the cavity explored.

In addition to the great number and variety of conditions which are well known as causes of intestinal obstruction, there are many which we would hardly con-

sider as common causes of acute ileus, yet they are causes, and they are purely surgical conditions, and can be dealt with in no other way. For example, a case of obstruction due to cyst of the ileum and cecum is reported by Hochenegg, and Magnoux reports a case of invagination due to the presence of a myoma. This author collected from the literature reports of eleven cases in which this condition was caused by myoma. Morison records a case of obstruction due to polypus of the small intestine.

Neoplasms of the intestine may suddenly produce symptoms of obstruction of the bowel without previous warning. It is probable that in such cases hypertrophy of the muscularis above the obstruction compensates for the narrowing of the lumen, but with the lodgement in the constricted segment of some unusually large or hard mass of intestinal contents, an acute fulminating ileus suddenly develops. The sudden appearance of ileus does not always mean that a recent lesion is the cause.

It has been said by Treves that in ileus too much prominence must not be given to the mere circumstance that the bowel is obstructed, that to be sure obstruction of the bowel is often the prominent symptom, but not the sole basis upon which the malady depends. It can hardly be denied that the issue of a given case will depend more or less directly on the length of time during which the intestine has been obstructed, regardless of whether the intestine be strangulated or plugged with its own contents. But mere plugging will not often produce the classical condition of ileus. The bowel may be completely plugged for weeks, and the patient make a good recovery. If, however, the intestinal wall be strangulated, its circulation blocked, the symptoms immediately become urgent. Here we must think of "strangled vessels," and beginning gangrene. Unfortunately, we too often confuse these cases of strangulation with mere obstipation or impaction.

It should be borne in mind that the condition of ileus

with strangulation of the bowel is very rarely produced by mere plugging, and that when the symptoms of genuine ileus appear, nine times in ten the time of usefulness of the syringe and the enema is gone. I would not place a low appreciation upon the colonic douche. Its proper use requires brains and art and patience, and in no case should radical surgical means be considered until the colonic douche has been faithfully tried, but as a rule lavage of the stomach, inversion of the patient and colonic injection of oil, air and water are at best only palliative. Capillary puncture, abdominal massage and the administration of opium are perilous and reprehensible in practically all cases. With prompt diagnosticating, prompt operating and better operating the mortality will be steadily lowered.

It may be said that, as a rule, the urgency is not so great in obstruction of the large intestine as in closure of the small gut. In the former sedatives, hot stupes, taxis and enemata may be justly employed for a longer time than in the latter.

To some it might seem unnecessary to urge the necessity for early operation in cases of intestinal obstruction with ominous symptoms. This excludes mere obstipation, for the reason that impaction *per se* does not produce severe symptoms, as before observed, but physicians, for the most part, are for some reason not recognizing intestinal strangulation promptly enough, except it be consequent upon sudden descent of a hernia, and are not promptly advising operation.

It is a dangerous surgeon who counsels operation merely for the sake of operating, and who would operate in a "stable with a pocket knife," but he who bears the name of surgeon, and who is adequately equipped, and can provide a reasonably aseptic environment, but who stands by with folded hands, resigning these cases to Providence and the rectal douche, is ten times more dangerous. The surgeon who does not realize the limitations of his art is a dangerous man indeed, and he who

is not aware of its possibilities is useless. But no man is more dangerous nor more useless in his community than the physician who would cling to a case of mechanical strangulating intestinal obstruction which he has been unable to accurately diagnosticate, and which he has not the facilities nor the intrepidity to treat in the only way which avails, that is by operation.

OCCURRENCE OF THE STRONGYLOIDES INTESTINALIS IN THE UNITED STATES.

MARSHALL LANGTON PRICE, M.D.
BALTIMORE.

I present for your consideration a somewhat brief and imperfect account of one of the rarer nematodes; rare, at least in this climate and latitude, though occurring with considerable frequency in tropical and sub-tropical countries. I refer to the *Strongyloides intestinalis*. The parasite is endemic in Cochin China, where it occurs in association with the diarrhea peculiar to the country—hence the name bestowed on it by European physicians, "Cochin China diarrhea."

This animal forms one of the class of parasites whose pathologic action is limited to the intestinal canal, and does not extend, as does that of the *Amœba coli,* to the liver and other organs. Teissier has claimed to have observed rhabditiform embryos circulating in the blood, and to have produced metastatic foci in the lungs of infected frogs. These observations, however, have not been confirmed by any other observer, and, for the present, we must conclude that the parasite's pathologic influence is limited to the gastrointestinal tract. The consideration of the diseases caused by animal parasites, particularly those infecting the intestinal canal, is especially opportune at this time, as the different varieties of acute and chronic diarrheas and dysenteries are only now beginning to emerge from the obscurity which has previously enveloped them, and are being placed on a rational basis, where the knowledge of them previously

was wholly crude and empirical. This knowledge has already borne valuable fruit; you are familiar with the work recently done under the direction of Professor Welch, of Baltimore, in the investigation of the summer diarrheas of infants, of the discovery of the essential cause, and of the preparation of a serum which promises a specific against a disease causing more than one-half the mortality in urban communities during the summer months.

It is only in the last decade that we have begun to realize that many cases of chronic intestinal flux in this latitude are due to the *Amœba coli,* and one form of parasite which we previously considered wholly indigenous to the tropics, has thus been given a wide sphere of activity in the diseases of temperate climates. We have later been able to recognize the cause of another class of diarrheas through the discovery by Shiga and Flexner of the *Bacillus dysenterica,* and it is probable that many other tropical parasites will be found to be the pathogenic factors of our native diseases. The rôle played by the strongyloides genus in diseases of the United States is hard to ascertain at the present time, but I do not believe it will ever be a prominent one in this climate, yet it will take a place as an etiologic factor in many individual instances in which the nature of the disease was previously unrecognized. The occurrence of the *Strongyloides intestinalis* in the United States was first reported by Thayer[1] from three cases occurring at the Johns Hopkins Hospital, Baltimore, his work remaining the best monograph published on the subject in the United States.

A short résumé of the history of the parasite may here be of interest:

In 1876, Normand, a medical officer of the French navy, while investigating the diarrhea among French troops returning from China, discovered this parasite in

1. Journal of Experimental Medicine, vol. vi, No. 1, Nov. 29, 1901.

the stools. It was studied by Bavey in the same year, and the latter named it *Anguillula stercoralis*. in analogy with the similar terrestial anguillula *Rhabditis terricola*, genus leptodera. The parasite is found constantly in the stools of these patients, and Bavey and Normand considered it the probable cause of Cochin China diarrhea. When these stools were kept warm for four or five days, sexual development took place, some of the parasites assuming the male and some the female type—the original first observed being, indeed, only an embryo. The following is Bavey's description of the sexually mature forms:

The length of the female is about 1 mm. fully grown, the breadth about 0.04 mm., body cylindrical, slightly diminishing in size anteriorly and tapering to a sharp point posteriorly. When the worm retracted forcibly, slight transverse furrows were to be seen. The mouth possessed three distinct lips and was continuous with the triangular esophagus, which after a stricture dilated again to a second ovoid enlargement. The intestine which followed was fairly visible and ended in a little protrusion on one side of the body near the base of the tail. The intestine was pushed slightly aside by the uterus. A little below the middle of the body on the ventral side opened the vulva leading to the uterus, which extended from the intestinal ventricle to a point near the intestinal opening; here the eggs were massed in varying numbers. In some instances the young had actually broken the shells and were free in the uterus, though more often the eggs on deposition contain well-formed motile embryos.

The male is about 1/5 smaller than the female; the peri-intestinal cells are more clearly outlined and were accompanied by another long gland, which seemed to consist of small rounded globules. This organ, doubtless the testicle, entered the base of the tail in two small horn-like spicules with tapering extremities curved inward; these spicules contained canals . . . coiled in the same direction . . . and were twice as short.

In the following year a complication occurred in the history of the parasite, with the discovery by Normand of a nematode in the small intestines of a patient dead of Cochin China diarrhea. This was similar to the parasite found in the stool, but larger than even the sexually differentiated forms, and possessing other

structural differences. Bavey was able to obtain it from several successive autopsies following death from this cause. He considered it a distinct species, which he called the *Anguillula intestinalis.* It was found only in the female form and was described as follows:

> Length 2.20 mm., average breadth 0.03 mm., the body little tapering anteriorly, terminated rather suddenly posteriorly in a conical tail, the extremity of which appeared rounded and even a trifle dilated . . . . the surface showed a delicate transverse striation; the mouth . . showed three small lips. It opened into an esophagus, practically cylindrical, which occupied about ¼ of the length of the animal, and showed neither swellings nor striations; it was followed by an intestine with which it might readily be confounded without a marked change of color. This extended almost to posterior extremity of the body, but it ceased to be visible in the middle part, which was occupied by a very large elongated ovary. The vulva was situated in the posterior third of the animal and in its neighborhood the uterus contained five or six elongated eggs . . the eggs and viscera were of a yellowish green color and rather opaque and apparently finely granular.

The parasitic form is found at autopsy in the small intestine. They are abundant in the duodenum, less numerous in the jejunum, and absent in the ileum. They seldom reach the stomach under normal conditions, but have been found in the vomitus after an emetic. In stools which have been kept warm for a varying period there is another embryonic form which is frequently seen; this is a much longer, thinner embryo of a more homogeneous constitution, and more actively motile than the more common rhabditiform embryo. This represents the strongyloid form in the embryonic state. The parthenogenetic female shows also the strongyloid type, and indeed, probably develops directly from the strongyloid embryo. The strongyloid embryo, although longer than the rhabditiform, never attains the size of the parthenogenetic mother worm; the average length is from 0.4 to 0.5 mm., the greatest length being 0.7 mm., while the parthenogenetic worm averages 2.20 mm. The following is a comparison of the size of the different varieties:

	Rhabditiform.	Filariform.	Free Sexually.	Partho-genetic, M. W.
Length	0.33 mm.	0.50 mm.	1. mm.	2.20 mm.
Breadth	0.022 mm.	0.02 mm.	0.04 mm.	0.03 mm.

The eggs are oval, and resemble uncinaria eggs, and measure 60 to 70 microns by 34 to 39 microns.

Following Normand's original investigations of the parasite as occurring in Cochin China, other observations were made on the parasite in the tropics. Laveran, Liebermann, Roux and Chartaing each reported cases in 1877 and 1878. Chauvin reported a case originating on the Island of Martinique. Grassi, in 1879, studied the worm, and placed it in a species which he called strongyloides, closely allied to the genus strongylus, of which the most important member pathologically is the uncinaria, or *Strongylus duodenale,* or, as it is called in Europe, *Anchylostoma duodenale,* from which it differs morphologically by certain structural peculiarities, and pathologically by important differences which will be described later. In 1880 the parasite first appeared in Europe with the report by Perroncito of its discovery in the intestinal tract of laborers in St. Gothard tunnel, in whom it occurred in association with the *Anchylostoma duodenale* in certain grave anemias described by Perroncito at that time; since then sporadic cases have been reported from Brazil, Russia, Sicily, Italy, Dutch Indies and Egypt. In 1899 cases were reported along the Rhine and in East Prussia. In 1901 Strong described cases occurring in the Philippine Islands.

In order to understand the divergent opinions that have been held in regard to the unity or heterogenicity of these nematodes it is necessary to describe the forms of parasites that may be found in the alvine discharges of the patients suffering from Cochin China diarrhea during life, or in their intestinal canal after death, all of which are now believed by the preponderance of observers to be morphologic variations of the same parasite. These are, briefly:

1. The rhabditiform embryo *Anguillula stercoralis,* found in the stools on immediate examination.

2. Filariform embryos usually found alone in the stools after one or two days, replacing the rhabditiform embryos.

3. The sexually differentiated form, *Rhabditis stercoralis,* occasionally found in the stools after a varying period—usually about five days.

4. The parthenogenetic mother worm, *Anguillula intestinalis,* found in the intestinal canal of the host at autopsy.

It appears that the worm shows a different development here from that which it exhibits in the tropics. The development of the sexually differentiated living generation must be regarded as the highest exponent of the parasite. This manner of development appears to occur only rarely in temperate zones, and in these regions we must consider that some conditions are less favorable to the parasite's attainment of the highest form, the difference lying either in intrinsic conditions affecting the culture medium, i. e., the stool in which it occurs, or in extrinsic causes affecting the surrounding atmosphere, or in conditions associated with both factors, such as heat, moisture and temperature. It may be premature or inexpedient to theorize as to the exact manner in which these various elements act, still we may presume that the difficulty lies rather in the culture medium than in the surrounding conditions, for while we can largely reproduce the extrinsic conditions, such as moisture, temperature, light, etc., we can not ever exactly reproduce the culture medium, for the latter in the tropics differs greatly from that of the temperate zones, both on account of the different foods which go to form it, and the different bacterial flora which it contains, the latter being much more varied and profuse in the tropics. In support of this view are Leichtenstern's recent observations (1899), that the sexually differentiated generation

is cultivated most frequently from cases recently imported from the tropics.

I will later call attention to the disappearance of the parasite from the stools synchronously with that of the numerous bacteria which the latter contains. This condition, while not an invariable rule, is a coincidence of much significance.

The parasite may be arrested at an intermediate stage of its attempt at sexual differentiation, and accordingly, we may find the nematode in our cultures in three distinct states:

(*a*) No attempt at evolution may be made, and the parasite occurs only as the rhabditiform embryo.

(*b*) The parasite may be able to attain the male development, but be unable to attain the female type, a few male parasites occurring in the stool.

(*c*) Complete sexual differentiation may occur, and both male and female types will be found.

Whatever further development may now occur if the parasites are ingested, if still remaining external to the body the filariform embryo is finally produced; in the first case by direct transformation of the rhabditiform embryos, in the last case by the interposition of another generation of sexually differentiated worms.

Keeping these features in mind, how are we to regard the filariform embryo in its relation to the other types of parasites? If we regard the free sexual form as the highest type; as the filariform variety follows after this, it appears that the filariform embryo must be considered an involution form from the highest type. The filariform embryo is, moreover, the last form found in the specimen before the final disappearance of the parasite from the stool. This disappearance must occur, even under the most favorable conditions, in a rather short period. It is probable that the strongyloides embryo is more adapted to the relatively unfavorable conditions found outside the body, and is, moreover, the usual form in which the intestinal canal

of the host is infected. If infection occurs in this way it is probable the filariform embryo becomes directly transformed into the parthenogenetic mother worm, which then commences to cast off its ova, and these, in turn, undergo development into the rhabditiform embryos. Leichtenstern has been able to demonstrate this process directly. He administered to human beings filariform larvæ which he had produced by the direct transformation of the rhabditiform variety. After seventeen days rhabditiform embryos were found in the patient's stools. In cultures these became transformed into the filariform variety, but a number also became sexually differentiated, forming a free living generation, the *Rhabditis stercoralis.*

Leuckart claims that the genesis of the parthenogenetic mother worm is the essential step in the regeneration of the rhabditiform embryo from the filariform. He says, "The structure of the filariform embryo is such that they can not possibly develop again into the rhabditis form. There must be another worm which develops from them, which in the shape of its body and the structure of the esophagus resembles these." The parthenogenetic worm, the so-called *Anguillula intestinalis,* appears to represent this intermediate form which Leuckart considers essential. Hence, while the rhabditiform embryo is transformed directly into the filariform, the filariform can not return to the rhabditiform variety without the aid of the intermediate form. The development of this parthenogenetic worm has never been noted outside of the living host. These conditions probably explain the ultimate disappearance of the parasite from the stools, when the latter are kept for a certain period, the parasites continuing their evolution until the filariform embryo is produced, and beyond this they can not go without the aid of a suitable host. The rhabditiform, as such, does not seem able to survive more than three or four days in the stools.

To trace the life history of the parasite on the assump-

tion of the unity of the various forms, we may conceive of a parasite entering the host as the filariform embryo; within the host becoming transformed into the parthenogenetic female, *Anguillula intestinalis,* the latter giving off numerous ova which, hatching rapidly before leaving the intestines, appear as the rhabditiform embryos (*Anguillula stercoralis*) in the discharges. The next transformation of the rhabditiform embryo depends on the environment. In the tropics the parasite becomes habitually sexually differentiated, forming the species known as the *Rhabditis stercoralis,* from the eggs of which are generated the rhabditiform embryos, which undergo final transformation into the filariform variety. In the temperate zones the rhabditiform embryo passes into the filariform without the interposition of the so-called *Rhabditis stercoralis.* In either event the filariform embryo is the final stage completing the cycle.

The first case in this country described of infection with the *Strongyloides intestinalis,* was that of a German tailor, aged 56, who entered the Johns Hopkins Hospital, Baltimore, Dec. 7, 1896. The case was complicated with intestinal infection with the *Trichomonas intestinalis* and amebic dysentery, which latter disease proved fatal on the development of an abscess of the liver. The parasite was discovered by Strong in the stools, and was reported, together with two like cases, by Thayer in 1901. Thayer's account of the case is summarized below:

CASE 1.—*History.*—K., a German tailor aged 52, was admitted to the Johns Hopkins Hospital Dec. 7, 1896, complaining of pain and swelling in the right side of the abdomen and over the lower ribs.

The family history was negative. He had had measles in childhood, but had otherwise been a healthy man. He denied venereal disease. Up to six years before he had lived in Austria.

Three years before entry he began to suffer from diarrhea, which has continued since that time. There were daily from two to five operations, which occasionally contained a little blood; these were never associated with pain. Three weeks before entry he began to notice a painful swelling on the right

side over the lower ribs. He had had no chills, and as far as he knew no fever. For two weeks before entry there had been a slight cough without expectoration.

Physical Examination.—The patient appeared emaciated and feeble. The heart and lungs were negative. The abdomen was

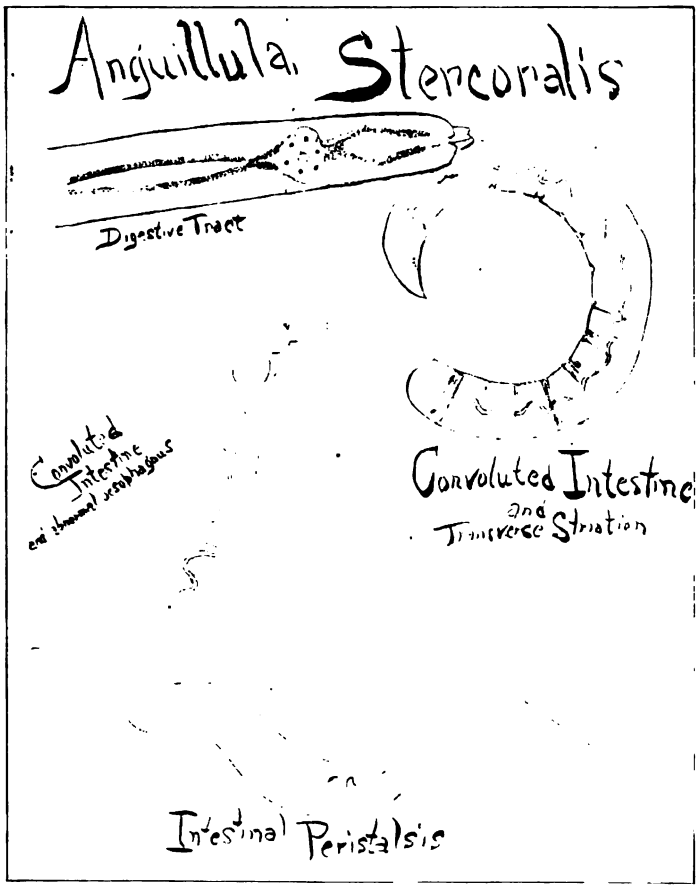


Fig. 1.—Details of the digestive tract and morphological variations—intestinal peristalsis (rhabditiform embryo).

somewhat distended; there was slight movable dulness in the flanks. On the right side over the cartilages of the tenth and eleventh ribs there was a large fluctuating swelling, 7½ cm. in diameter; no tenderness or redness. Between this and the

spinal column there was another swelling, about 15 cm. in diameter, extending from the median line to the spinous processes, and from the twelfth rib over the crest of the ilium downward for a distance of 7 cm. The skin over this was adherent, red and thin. The hepatic flatness was not increased.

Operation.—Immediately after entrance this abscess was incised and evacuated. The cavity contained a large amount of puriform material with dark shreds and considerable grayish debris. The cavities extended into the liver. About one and a half pints of fluid were evacuated; this contained many large, actively motile amebæ, showing the ordinary characteristics of the *Amœba coli.*

The patient had daily from two to four fluid, brownish, foul-smelling stools, which contained besides *Amœba coli*, considerable numbers of the *Trichomonas intestinalis*, as well as numerous small nematodes, identified by Mr. Strong as the rhabditiform embryos of the *Strongyloides intestinalis.* The wound and the colon were irrigated with a solution of quinin, 1/1000. The patient gradually failed, and died on December 26.

Autopsy.—The protocol of the autopsy reads: Amebic dysentry; ulceration of the large intestine; retroperitoneal abscess; operation wounds; parenchymatous and fatty degeneration of the kidneys; fatty liver; fatty heart; intestinal infection with *Trichomonas intestinalis* and *Strongyloides intestinalis.*

The large intestine showed the extensive ulcerations due to the amebic infection; the small intestine was free from ulceration, but showed slight occasional ecchymotic patches.

Along the whole of the small intestine Dr. Stiles and Mr. Strong found embryos of the *Strongyloides intestinalis;* and parthenogenetic mother worms were found in the duodenum and jejunum.

Dr. Strong was successful in this case in the cultivation of a single sexually differentiated parasite. This was of the male type, and was dead when discovered. No female forms were found.

The second case did not occur until nearly three years later. The symptoms of this case were chronic diarrhea and emaciation, and were not especially marked, and might easily have escaped the more superficial examinations, as it was only observed as a dispensary case. The case did not escape the vigilance of Dr. Thayer, in whose hands it fell, and the diagnosis was made almost as soon as observed.

CASE 2.—*History.*—A newsboy from Richmond, Va., where he had lived all his life. The family and previous history showed nothing bearing on the case. He had had the usual diseases of childhood, and five years ago had tertian ague.

Three years before entry the patient began to suffer from

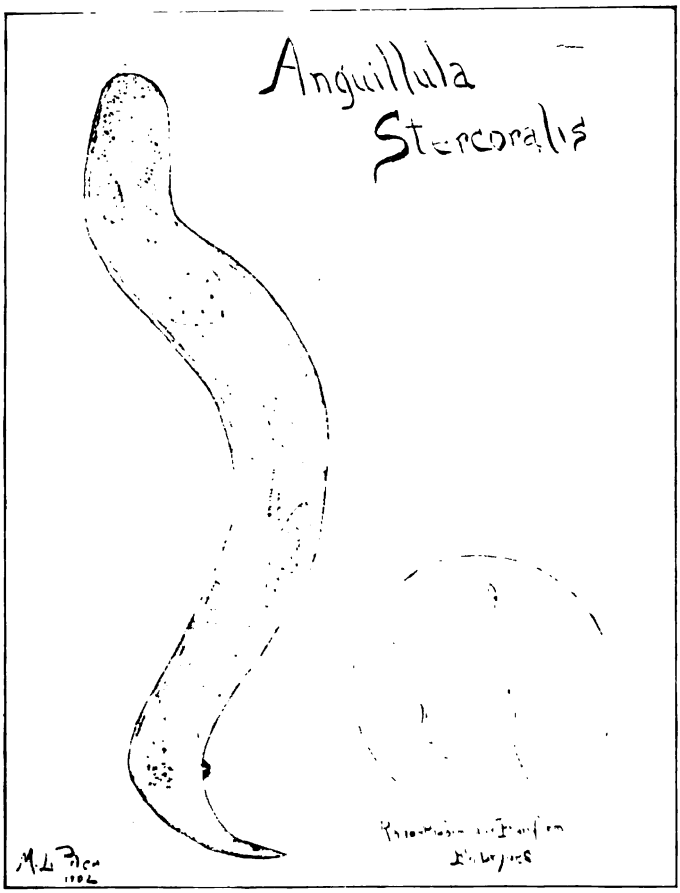


Fig. 2.—Rhabditiform embryo. Semidiagrammatic to show structure.

diarrhea, the attack coming on during the summer. The operations were very thin and yellowish in color, containing at times a small amount of blood, though this had not been noticed for a year and a half. The passages occurred mainly at night, as many as seven or eight in 24 hours. but of late

only about four. There was at first considerable mucus in the stools, but this had been practically absent during the two years before entry. The diarrhea had never been associated with any pain. The patient had been, however, growing gradually weaker, and during the second year of his illness he had been compelled on one occasion to take to bed for two weeks. Four months before entry he gave up work on account of weakness, though up to that time he had felt tolerably well. The appetite was good. There were no dyspeptic symptoms.

Physical Examination.—A slight emaciated boy of sallow color. The lips and mucous membranes were somewhat pale; tongue clean. Examination of the heart and lungs was negative. The abdomen was not distended. The spleen was not palpable. No enlargement of the liver.

The patient entered the hospital July 7 and was put to bed and given a liquid diet. The only medication given at first was bismuth. Later the patient was, on two occasions, given thymol gm. 2 (gr. xxx), the dose being repeated after an interval of one hour, and following this an active purge. Male fern was given in like manner in doses of one dram.

The patient's symptoms improved greatly and he gained twenty pounds in weight. He was discharged at his own request August 26. The stools on discharge still contained moderate numbers of the rhabditiform embryos of the parasite.

CASE 3.—*History.*—A native of Maryland, who had never been out of the state except on an occasional visit to Washington. J. S., a farmer, was admitted to the Johns Hopkins Hospital July 19, 1900, complaining of diarrhea and swelling of the feet. The patient had been a farmer all his life; his habits have been good; he has had no serious illnesses except chills and fever fifteen years before entry. He had considered himself a healthy man up to six months ago, when he began to suffer from diarrhea and progressive weakness. The stools, very frequent and sometimes involuntary, were fluid, but never contained blood. They were small in quantity. During the two months before entry the patient had on several occasions vomited a yellowish fluid. For two days he had had sharp, griping pains in the lower abdomen. For a month there has been edema of the feet and legs. . . . During the last two months he has lost considerably in weight and much in strength.

Physical Examination.—Moderate atheroma of the radial, slight abdominal distension and flatness in the flanks. On examination a moderate number of characteristic rhabditiform embryos of the *Strongyloides intestinalis* were found, together with a few red blood corpuscles and large numbers of leucocytes. The temperature was slightly elevated, 100.6 F., on entry, but with the exception of an occasional rise to a point

between 99 and 100 it remained subnormal during the greater part of the patient's sojourn in the hospital.

On entry the patient was given a milk diet and powders of tannogen and bismuth subnitrate, q. 4 hours . . . . on April 12 . . . he was given thymol, gr. v. q. 4 hours . . . . on

Fig. 3.—Parthenogenetic mother worm (parasitic form).

August 13 the thymol was discontinued and four doses of santonin were given at hourly intervals. . . . the patient was considerably better. There was a gain of 21½ pounds in weight between July 29 and September 17, the date of his discharge.

The blood examination from all three of these cases showed moderate anemia, the hemoglobin in the first case of Thayer's series registered 48 per cent., and in Case 3 of this series 57 per cent.; in both of these cases, however, there were complications present; the first case was complicated by amebic dysentery, the last case with a probable renal sclerosis. The high leucocyte count observed in the first case is easily accounted for by the amebic infection. The presence of a hyperleucocytosis in Case 3 is hard to account for; the one count made showed 21,500 leucocytes to the cubic mm.; unfortunately, no further count was made. None of the three cases showed any eosinophilia. The differential count in the first case gave:

Polymorphonuclear leucocytes	88.3 per cent.
Small mononuclear leucocytes	7.5 per cent.
Large mononuclear leucocytes	2.4 per cent.
Transitional forms	1 per cent.
Eosinophiles	.3 per cent.

The blood count showed 3,412,000 r.b.c., leucocytes 32,100; hemoglobin 48 per cent. Twelve days later the eosinophiles had fallen to 0.1 per cent., and the leucocytes to 30,000. The *valeur globulaire* was little affected, the blood generally showing the changes similar to all secondary anemias resulting from loss of blood.

Thayer's second case is of much interest on account of the occurrence of a number of eggs in the stools. This is very rarely observed in infection with the *Strongyloides intestinalis* unless there also be present an uncinarial infection.

After the report of Thayer's cases no further cases were observed until the middle of 1902, when two cases were discovered within a few months of each other; one occurring in the practice of Dr. Moore at Galveston, Texas, the other case occurring at the University Hospital in Baltimore in my own service. This case I wish to describe in some detail:

CASE 4.—*History.*—Entered the University Hospital July 25, 1902, complaining mainly of a chronic diarrhea, which had lasted about five months. The movements varied from

about 2 to 15 daily; average was about 10 to 12. Operations were painless and were not accompanied by tenesmus. Alvine discharges were watery and contained mucus. There were occasional slight griping pains in the abdominal region. Patient had lost about 15 pounds in the last three months. Occasional sweating was present, both at night and during the day and accompanying this mild rigors. There had been slight cough lasting about two years, and occasionally sharp pains have been felt in the chest. The patient was operated on

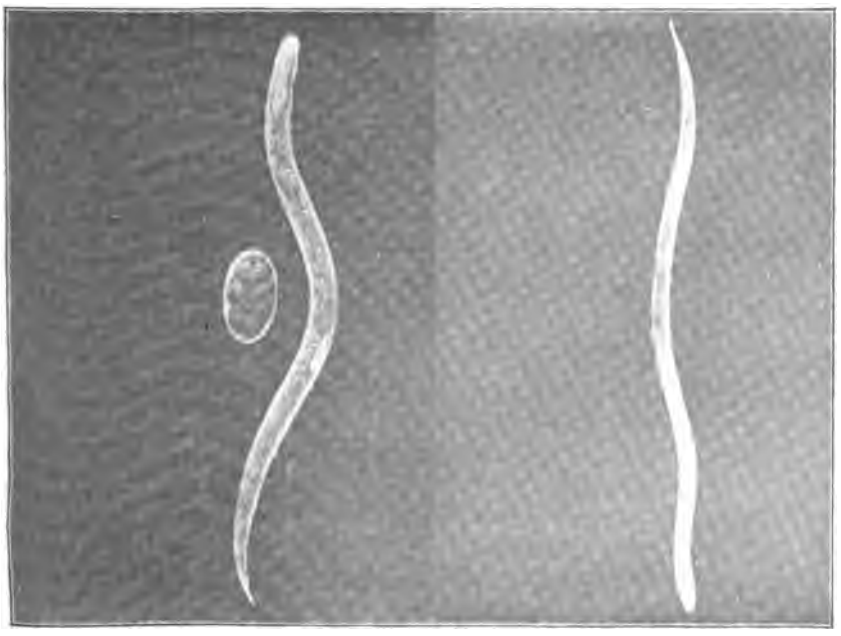


Fig. 4.—Embryos. A. Egg. B. Rhabditiform embryo. C. Filariform embryo.

about two years ago for fistula in ano. He lost two sisters from pulmonary tuberculosis, and his father died of the same disease at the age of 20.

Physical Examination.—Patient is a well developed man, rather thick set, of excellent physique with a clear, ruddy complexion, but with some slight pallor of the mucous membranes. Height 5 ft. 6 in. Weight about 135 lbs. His previous regular weight has been about 145 lbs.

Eyes.—Clear and bright, pupils react readily to light and accommodation. Palpebral mucous membrane a trifle pale.

Lips showed some pallor and a faint cyanosis. Tongue was pink, moist and clean . . . . moderate enlargement of tonsils . . . no other glandular enlargement. . . .

Chest.—The thorax is large and well formed, costal angle about 45 degrees, expansion fair, equal on both sides. Both supra- and infraclavicular fossæ are well filled. Apices of the lungs rise about ¼ inch above the clavicles. There is good resonance over both clavicles and over the anterior thorax. Expiration a trifle prolonged . . . a few sonorous râles were heard . . . at the right apex posteriorly the respiratory murmur was roughened and the sonorous râles more numerous and an occasional clicking sound was heard. The voice sounds are considerably exaggerated. On percussion there is slight diminution of resonance and a slightly elevated pitch.

Heart.—Situation of the apex beat is normal, cardiac sound is clear, both at the apex and base.

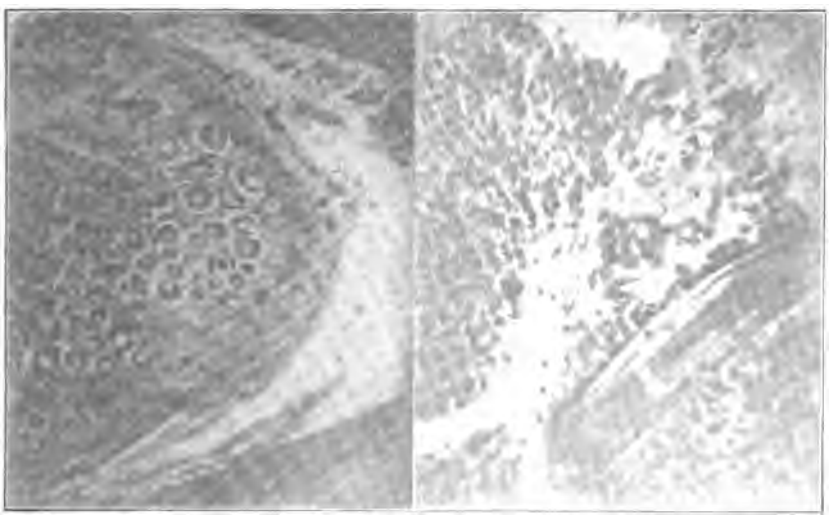
Abdomen.—Hepatic flatness begins at the sixth rib and extends about ⅛ inch below the costal margin. The liver and spleen are not palpable. Abdomen is soft, no tenderness or tympanites. Lymphatics somewhat enlarged. . . . .

There were several possible conditions to be considered as the essential cause of this diarrhea. The tubercular history with the history of fistula, the occurrence of slight rigors and sweating with cough lasting two years, rather indicated a tuberculous process with ulceration of the bowel. The excellent physical condition of the patient, absence of emaciation and of glandular or peritoneal involvement were at once sufficient, however, to negative this assumption. It is seldom that extensive tubercular ulceration lasts more than a few months without some local or general peritoneal involvement. The long periods of remission are also antagonistic to this assumption. The absence of pain, the long remissions and liver enlargement, together with slight rigors, suggested an amebic infection. On examination of the first specimen of stool, however, a single parasite was observed. This parasite was very sluggish and its movements only lasted about half an hour, and owing to this fact, and to the examination being made at night, it was not possible to determine accurately its characteristics, but it appeared to be an uncinaria.

Examination of the Stools July 29.—Stool of semi-solid consistency, brown in color, odor not especially marked.

Microscopic Examination.—Red blood and pus cells abundant—fine granular detritis—few starch granules (in addition to the parasite already described).

Treatment.—The next day the case was seen in consultation with Drs. Thayer and Atkinson, and on passage of the rectal tube Dr. Thayer was able to find a number of the rhabditiform embryos of the *Strongyloides intestinalis.* The patient was put on liquid diet for twenty-four hours, and then on soft diet, and was confined to bed. The only medication given was small doses of quinin. Under rest and diet the symptoms



A B

Fig. 5.—Section of duodenum through the crypts of Lieberkuhn. A. Showing cyst formation in crypts. B. Section of embryo in crypt with surrounding infiltration and karyokinesis.

abated greatly. The number of daily movements averaged about the same, but the involuntary evacuations of the bowels at night ceased.

Examination of the Stools August 2.—Granular detritis, pus and blood cells diminished; numerous motile and non-motile bacteria.

The stools were now examined daily. The occurrence of the parasites was very variable; occasionally the fecal drop would contain five or six specimens and at other times none would be found after a careful search through a number of specimens

lasting two hours. The greatest number found in any drop of fecal matter did not exceed ten or fifteen. There appeared to be a constant relation between the number of parasites and the number of bacteria occurring in the stool. When there were no bacteria evident, on microscopic examination, the search for parasites proved futile; on the other hand, stools containing large numbers of bacteria usually contained numerous parasites. Nearly all the softer stools contained both bacteria and parasites. The stools, both microscopically and macroscopically, showed the presence of an intestinal indigestion. They contained during all of the diarrheal relapses undigested food, and the odor was usually offensive. In one instance fermentation had occurred, and bubbles of sulphuretted hydrogen were being given off rapidly. This specimen contained numbers of parasites.

A certain technic is necessary in the examination of the stools, and in the cultivation of the other forms of the nematode, especially if the endeavor is to be made to cultivate the sexually differentiated forms.

The stools as soon as passed should be placed in open vessels and kept at room temperature. It is best to divide the stool into three portions so as to observe the parasite under varying conditions. One specimen may be placed on the top of the sterilizer, one beneath and one at room temperature. A little normal saline solution may be added to the stools of the more consistent kind, the embryos having a tendency to collect in this portion. For examination, a considerable dilution of the drop is always necessary, otherwise the embryo, being almost transparent, will escape observation. It is best to commence the examination with the ¾-inch objective, and reduce the light somewhat.

Examination of the Blood August 10.—An examination of the blood was made August 10 with the following result:

Hemoglobin	82 per cent.
R. B. C	4,672,000
Leucocytes	7,800
V. G	0.88

A marked eosinophilia is present.

This moderate anemia gradually improved with the improvement in the general condition. The leucocyte count was practically normal, and never at any time showed a marked increase.

An eosinophilia was present sufficiently marked to be plainly evident on the examination of the fresh blood. Eosinophilia is not a symptom generally observed in intestinal infection with the *Strongyloides intestinalis*, and its presence in this case suggested strongly a condition of mixed infection frequently observed, viz., where the strongyloides and uncinaria are cohabiters of the intestinal canal. This condition strengthens the possibility that the first specimen observed was really an uncinaria. The eosinophilia in these cases frequently persists after the anemia and other symptoms have disappeared and the parasite has apparently been completely eradicated. A differential count was made some days later by Dr. Rankin, resident pathologist to the University Hospital, and he examined several stained blood smears at the same time:

QUANTITATIVE:

R. B. C.	4,736,000
Leucocytes	7,860

DIFFERENTIAL COUNT, LEUCOCYTES:

Polymorphonuclear	250	47.5 per cent.
Small mononuclear	157	29.6 per cent.
Large mononuclear	50	9.5 per cent.
Transitional	8	1.1 per cent.
Eosinophiles	61	11 per cent.
Cells counted	525	

Qualitative.—Red blood corpuscles show little change. No poikilocytosis and corpuscles uniform in size. Slight pallor. White blood corpuscles, polymorphonuclear leucocytes, show every variety of degeneration. (*a*) There is increase in eosin staining. (*b*) There is loss of neutrophilic granules. (*c*) There is vacuolization, fragmentation and loss of outline in the body of the cell. (*d*) Nucleus is enlarged, paler and shows clear vacuoles—hydropic degeneration. (*e*) Perinuclear basophilia marked. The nuclei also show supernumerary lobes in many cells. Small mononuclears vary considerably in size, are more granular and the nucleus often irregular. Large mononuclears much increased, show marked degenerative changes, principally in an increase in granules and considerable hydropic degeneration. The nuclei of many of these cells are much fragmented and many look as if they were undergoing karyokinesis. Eosinophiles vary considerably in size and in number of granules contained; but all the granules are uniform in size and of equal staining power. No iodophilia.

On August 21, commencing at 11 a. m., the patient was given thymol, grs. vii, every five minutes until twelve doses were given, the patient receiving in the hour gm. 12 (180 grains).

The patient had some abdominal pain and griping, not enough to give him any serious inconvenience, and experienced some slight nausea. He was freely purged, and eight speci-

mens of the stools were obtained, containing an abundance of undigested food. Any further purgation was considered unnecessary. The patient was given no food for twenty-four hours previously. The specimens of stool contained numbers of embryos, all of which were dead. No specimens of the parthenogenetic worm were observed.

One week later the same treatment was repeated, thymol being again given in heavy doses and following this a purge. In the evening an enema of sol. of quinin was given (1 to 1000).

After this treatment the patient improved considerably and was able to resume house diet. Examination of the stools, however, showed actively motile rhabditiform embryos, though in diminished numbers. The patient was now much better, and during my absence from the hospital he was discharged.

He disappeared from view until the beginning of April of this year. He had gone over to Washington, where he sometimes lived, and experienced a severe relapse. He gave his history in one of the Washington hospitals, and learning that he had been treated for a "foreign worm" a specimen of the stool was sent to Dr. Stiles, who found numbers of embryos in it. Since then Dr. Stiles has made systematic examinations of the stools in about 150 cases, and has succeeded in adding to our knowledge of the occurrence of the parasite in this country, five additional cases.

RHABDITIFORM EMBRYOS.

As the parasite occurs in the stools as the rhabditiform embryo, and probably never in any form (excepting rarely after a purge, when the passage of the parthenogenetic mother worm has been reported by one or two observers), it follows that the thorough understanding of this form is most essential to diagnosis; and I have, therefore, described it below at some length.

In a properly prepared specimen the embryo appears as a clear, almost hyaline, nematode, completely transparent and possessed of a very active motility, swimming among the granular detritus, and pushing aside the smaller obstacles in its path, and turning aside to pass the larger ones, when its progress is arrested by an obstacle of considerable size. The consistency of the nematode seems scarcely to exceed that of protoplasm, the parasite quivering like jelly with every movement, or when the microscopic stage was jarred or shaken.

It appears to possess a considerable degree of irritability, and when aggregated masses of bacteria would lodge against its side, motions of the tail would be produced toward the offending object.

The structure appears granular. The parasite, on examination, is seen to consist of two portions, an outer clear portion or envelope, and an inner granular zone. The outer portion, forming about one-tenth of the transverse diameter of the parasite, is clear and homogeneous, with the exception of a few fine granules. The innêr portion is markedly granular and is well organized.

With the death of the parasite the clear homogeneous structure is lost, the granules become larger and coarser and invade the outer envelope. Finally the whole parasite becomes uniformly granular and shrunken, its transparency is largely lost, and all distinction between the envelope and inner granular zone disappears. The parasite appears to undergo a rapidly granular degeneration as soon as dead.

The length of the nematode varies. The average is probably about 0.3 mm., the breadth 0.04 mm. The head is blunt and slightly conical in shape, and contains the anterior portion of the digestive canal. The body gradually enlarges from the head to its middle part, and then gradually diminishes in size to the thick and somewhat pointed tail. The head is largely occupied by the triangular or fusiform darker area of cells surrounding the digestive canal which passes from the cephalic extremity backward. This darker area becomes constricted as soon as the neck is reached, and after running for a variable distance in a uniform size, again expands into an ovoid or spherical area; leaving this, the canal again passes toward the caudal extremity. A slight fusiform dilatation occurs at about the middle of the body, the canal then passes on in its normal size until it reaches the base of the tail, where a saccular dilatation appears. This appearance, which has not been previously described, can be demonstrated in the majority of speci-

mens by careful examination, and no specimen in which the parasites were numerous failed to show one or more in which this structure was evident. The intestine, much diminished in caliber, turns abruptly after leaving this inferior dilatation and passes to the side of the animal, where it terminates in a slight mammilliform projection. Just below the middle of the body, the intestine was pushed somewhat to one side by a large oval body, quite refractile and containing rather large granules. This represents the undifferentiated sexual gland. The intestinal contents are plainly visible in the intestinal canal and consist of fluid containing numerous small particles in suspension. Among these can be seen dead bacteria and portions of bacteria and the ordinary granular detritus found in the stools. The motion of the parasite aids the passage of the intestinal contents toward the caudal extremity, but peristalsis has been provided for in another way, to be later described. The motion of the parasite is constant and never ceases, except momentarily; the motion, indeed, seems necessary to the life of the parasite, and when it ceases the parasite soon shrinks and undergoes the granular degeneration already described. The motion of the parasite apparently acts in somewhat the same manner as the cardiovascular system in higher animals in promoting the interchange of the tissue juices.

The minute structure of the digestive system shows many interesting details. The canal begins, in many specimens, in a procephalic projection, which has a cup-shaped depression on its anterior end. The digestive canal passes through this portion, which is transparent and non-granular, and not being especially refractile, is difficult to demonstrate in all specimens. The canal, passing into the head, enters the anterior esophageal expansion, which consists of stratified squamous cells with oval or flattened nuclei. It then passes toward the tail until it reaches the second globular expansion, where it is suddenly lost, plunging, in fact, into the center of

this mass. . This globular mass is made up of large secreting cells containing fine granules, and a large centrally-placed nucleus. This organ appears analogous to a stomach, and probably furnishes the digestive juices. In the center appears a round, clear area, which is the cavity of the organ showing through the cell mass. It is in this situation that the tridentate chitinous armature described by certain authors makes its appearance. I have never been able to demonstrate any opening in this situation, and have never observed the ingestion or excretion of any of the intestinal contents by means of it. The appearance is like that of the small Greek letter gamma. (The upper limb appears to be formed by the intestinal canal showing through the cells, and the two lower limbs by the interval between two of the large secreting cells, whose angle corresponds to the angle of refraction of the parasite.) As soon as this secreting organ becomes continuous with the intestinal canal (as occurs in the filariform embryo) this appearance at once is lost. About the middle of the body the canal enlarges slightly and extends some distance downward before it again contracts. This portion of the canal is lined by distinct columnar cells with dark nuclei; this appears in analogy with the secreting organ to be the absorptive portion of the parasite.

The digestive canal may at times be seen to suddenly contract longitudinally. The result of this is that a portion of the canal becomes convoluted or S-shaped, and its lumen in this portion becomes narrowed, or even obliterated; acting in this way as an adjunct to the more constant peristalsis produced by the movements of the animal. The effect of the globular dilatation seen at the caudal extremity is presumably to delay the intestinal contents until they can be effectually acted on by the tissue juices.

MORPHOLOGIC VARIATIONS OF RHABDITIFORM EMBRYOS.

The variations of form of the *Strongyloides intestinalis* do not end with the four distinct types mentioned,

but the embryonic forms, especially, show a number of other morphologic variations. It is important to realize this fact, as these variations may otherwise be mistaken for different species. Between the two widely varying types of embryos, viz., the filariform and rhabditiform, are found many intermediate forms. The youngest form of embryo is short and thick, the older specimens become longer and thinner, and in the shape of their bodies come to resemble the filariform type. The line of demarcation is, however, distinct between the two types in the structure of their internal organs. In one specimen observed both the filariform and rhabditiform embryos were found in the same specimen with others that in the shape of the body resembled the filariform, but were rhabditiform embryos. No marked variations occur in the filariform type, but variations in the rhabditiform embryos were frequent and occasionally marked.

In some of the embryos the intestinal canal was convoluted throughout. This occurred probably in less than 1 per cent. of the embryos observed, but in a single specimen examined two such were found. The esophagus showed a number of variations. Normally, the first esophageal enlargement was separated from the second by a considerable interval, the two portions being connected by the intermediate intestine. In one specimen, however, the interval was absent, the second expansion appearing at the lower end of the first. In this, as in a few other examples, the cephalic portion of the esophagus showed a double expansion.

In a few instances the embryos presented dark transverse striæ, alternating with lighter areas, the appearance being somewhat like that of voluntary muscle fiber. In these specimens the intestinal canal is usually convoluted, and along the outer and inner borders of the parasite appeared—corresponding to the origin of the dark bands—indentations, which became marked along the inner border when the parasite coiled.

It is probably an appearance similar to this which

Strong describes as two longitudinal rows of muscle plates placed along either side of the central canal of the older rhabditiform embryos.

FILARIFORM EMBRYO.

The filariform embryo is about double the length of the younger rhabditiform variety; the capsule or ectodermic layer is thinner and more distinctly outlined than in the rhabditiform variety, and is more perfectly hyaline, containing only a few scattered granules. The inner granular portion is uniform in width. The two expansions seen toward the cephalic end of the rhabditiform embryo have disappeared; the oval sexual gland can not be distinguished; the inner portion is made up of round refractive granules like those of the rhabditiform variety, but larger in size; the intestinal canal ends relatively nearer the tail. The parasite moves very rapidly with a graceful undulatory motion.

CLINICAL HISTORY OF THE DISEASE: ETIOLOGY.

The etiologic factors other than the parasite have not been as yet thoroughly studied. Infection usually takes place by the mouth. It is probable that the parasite is usually introduced in this way as the filariform embryo. Leichtenstern caused infection experimentally in this way by administering filariform embryos to a human subject. Infection has been produced in animals by administering the eggs. The vehicle by which the parasites enter the system is frequently, no doubt, drinking water, but this does not appear to be the only means. Normand during his stay in China drank only water imported from Europe in order to avoid infection, yet he suffered from a rather severe attack. Fresh vegetables, he thinks, may be a means of conveyance of infection, owing to the Chinese method of fertilization of land by human manure. Probably domestic fowls have a great deal to do with spreading infection, no doubt, through the mediation of fresh vegetables. Fowls are more susceptible to this disease than man, and the infection passes

readily from man to animal, and may pass with equal facility from animal to man. Infection through the skin has been demonstrated in rabbits recently under Dr. Stile's supervision. For human infection the infective material must be of comparatively recent origin, as the parasite does not survive long external to the body, even under the most favorable circumstances. The influence of sex in this disease has not been carefully studied. All of the cases which have come under my knowledge in this country have occurred in men. The occurrence of other diseases and of local disturbances of function in the intestine seem to influence, if not the actual infection, at least the manifestation of its results. The frequency with which the disease is associated with uncinariasis, with amebic dysentery, and with other diseases, seems to be more than a coincidence. Climate influences, as already stated, both the frequency of infection and the severity of its results. The more severe forms of the disease are infrequent in temperate countries.

PATHOLOGY.

The pathology of the parasite is mainly taken from Thayer's fatal case and from Strong's later studies. The changes found in the intestine are sufficient to refute the views of those observers who claim that the parasite is innocent to man. The possible ways in which the parasite may produce harm are numerous. Calmette's supposition that the parasite may give rise to substances acting as chemical irritants assumes some probability in the occurrence of a small round-celled infiltration about the ova and parasites, in the crypts of Lieberkuhn. The reflex effects may be a most important pathogenic element; certainly the constant intestinal indigestion, with its attending diarrhea and malnutrition, can not be easily explained by the small amount of inflammation present. The sudden involuntary passages are certainly a reflex nervous phenomenon. We already recognize that the presence of *Oxyuris vermicularis* in the mucous membrane of the rectum is capable of producing in chil-

dren many reflex disturbances, and it seems reasonable to suppose that the presence of a like irritant in the duodenum and jejunum can produce even more profound effects. Possibly some of the nervous phenomena observed by Sylvestri, such as vertigo, weakness and gastric disturbance occurring in connection with this parasite, may have been of reflex origin.

From Case 1 of Thayer's series Strong prepared sections of the intestine from which he was able to show the injurious influence of the parasite in the intestine. The microscopic appearances were both interesting and instructive. Both embryos, mother worms and eggs were found in the crypts of Lieberkuhn; no eosinophilia was observed about the eggs containing the crypts, but there was present a small round-celled infiltration and karyokinetic figures were observed in the surrounding epithelial cell. The following is a detailed description of the sections prepared by Strong:

Section 1.—Many eggs in various stages of segmentation, some containing embryos, seen generally lying in the bottom of the crypts of Lieberkuhn. In these crypts the epithelium is much compressed and often atrophied. The nucleus lies at the base of the cells, and about it no cell substance can be distinguished, as a rule. Some contain two eggs, and in such cases the epithelial cells seem markedly compressed and atrophied. About the crypts which contain eggs, particularly when they contain eggs which are segmenting or containing embryos not yet hatched, there are a few round cells about the crypt . . . In several sections where the egg has burst and the embryo escaped the epithelium of one side of the crypt has disappeared. About the crypt there is some proliferation, as is indicated by the karyokinetic figures seen in the nuclei of the surrounding epithelial cells . . . the underlying solitary follicles are somewhat swollen. In other crypts the egg rests on the basement membrane of the gland, the epithelium having (apparently) disappeared.

Not infrequently what appears (in transverse sections) to be small cysts are seen lying in the walls of the crypt, resting on the basement membrane. Apparently the eggs have disappeared from the crypts, leaving clear areas. There is no apparent infiltration with round cells about these so-called cysts.

Section 2.—Pieces of the adult female cut longitudinally and transversely. Not infrequently portions of the female

containing the vulva may be seen just penetrating into the lumen of the crypts, while others are lying longitudinally or cut transversely in the bottom of the crypt. Occasionally slight round-celled infiltration is noted at the bottom of the crypts containing parasites, and the solitary follicles are slightly swollen. . . .

Section 3.—**Many of the crypts contain embryos. In these crypts there is generally flattening and atrophy of the epithelial cells . . . the embryos appear to compress the epithelium . . . mitotic figures and small round cells are seen without the crypt. In several sections embryos are apparently bursting out of the crypt and have desquamated epithelial cells lying about them. One section shows an embryo beneath the epithelium of the villus.**

Teissier, in 1895, reported a remarkable case, in which the embryos of the *Strongyloides intestinalis* apparently appeared in both the blood and stools. On account of certain differences in his description of the parasite, and in the observations which he made on it, Thayer concludes that he was dealing with a parasite distinct from the strongyloides group. These differences were mainly as follows: (*a*) The finding of sexually differentiated forms in the fresh dejecta, as well as in culture specimens. (*b*) No internal structure could be demonstrated in the embryonic forms seen in the blood and the smaller forms found in the stools. (*c*) But one spicula was noted in the male form.

In addition to these differences, Teissier, in inoculating experiments on frogs, succeeded in producing metastatic foci in the intestine and lungs, which latter contained giant forms.

After discussing these differences, Thayer advances the supposition that the form of parasite with which Teissier was dealing was the *Ascaris nigrovenosa,* which corresponds closely to Teissier's description, especially in the occurrence of giant forms in frogs.

SYMPTOMS.

The symptoms are those of a more or less continuous diarrhea, characterized by periods of remission, which sometimes exceed one or more weeks' duration. The

onset is gradual, and it is some days, or even weeks, before the diarrhea is fully established. The presence of the parasite may be attended by no symptoms for an indefinite period, and none may ever present themselves. As the diarrhea becomes fully established, the evidences of intestinal indigestion appear; certain articles of food will not be digested or absorbed in the intestinal canal. Reflex gastric disturbances may occur; there is occasional heaviness and eructations and slight nausea; vomiting occurs as a symptom in some cases; usually only the severer ones. During the height of the attack the movements increase from twelve to fifteen, or twenty-five per day.

A striking characteristic of the diarrhea is the absence of pain, tenesmus, or any violent depression, such as occurs in other intestinal diseases attended by frequent and copious discharges. Mucus occasionally occurs in the stools, but is always small in amount; in a few cases a trace of blood is present at the onset. The diarrhea may suddenly assume a dysenteric or choleriform character, with purging, vomiting, cyanosis and collapse. This is in many instances due to the concurrent dysentery, to which persons suffering from this disease have a marked predisposition.

Another striking feature is the absence of any elevation of temperature in uncomplicated cases. With the onset of intercurrent disease, and with advanced malnutrition, an elevation of temperature may be present, but with patients otherwise healthy the temperature curve remains uniform during the most severe attacks. In the case observed in the University Hospital the temperature curve showed remarkably few fluctuations, never rising above 99 F. during the patient's stay of nearly six weeks in the hospital. On several occasions of acute exacerbation where the bowel movements exceeded fifteen daily, the temperature did not rise one-half of a degree.

Owing to the intestinal indigestion and consequent

malassimilation, there is progressive loss of weight during the acute periods; sweating is an occasional symptom, and a slight reflex cough is occasionally noted. In severe cases emaciation is advanced, there is great debility and weakness, and an intercurrent dysentery or other acute disease proves fatal. A striking and troublesome symptom not infrequently observed is incontinence of the bowel; this occurs most often during the night, but may occur suddenly in the day time. The desire to evacuate the bowels is sudden and almost irresistible. The tongue is usually clean and moist and the breath free from odor; there is no pain in the stomach, but occasionally some intestinal griping. The appetite is apt to be good, except in severe cases accompanied by nausea. The skin and eyes are usually clear, though the latter are occasionally subicteroid. Nervous symptoms are infrequent, but have been reported in a few cases. The symptoms observed were dizziness and vertigo with loss of appetite, weakness and slight nausea.

The blood changes are not usually marked; slight anemia is present, never approaching the degree observed in ankylostomiasis. Eosinophilia is probably not a symptom of uncomplicated cases; there is no leucocytosis. The stools present the ordinary appearance of intestinal indigestion, and contain undigested food. On microscopic examination there are present starch, granules and oil globules, together with pus, epithelial and red blood cells. After the disease is established the stools do not regain their normal consistence, but vary from a pasty to a fluid state. As would naturally be supposed, indigestible articles increase the diarrhea; corn especially, and occasionally beer, appear to excite a relapse.

DIAGNOSIS.

Diagnosis depends on examination of stools. The clinical features, however, are very suggestive; the occurrence of considerable intervals of remission, during which the patient largely regains his previous health,

the absence of pain or any elevation of temperature, and the absence of blood or mucus in the stools in appreciable amounts, are all symptoms individual to this disease. Incontinence of the bowel, if present, is extremely characteristic. In practice, amebic dysentery will be most frequently confused with this disease, as they also run a very intermittent course, and the elevation of temperature may be slight or absent. An examination of the blood in these cases will at once clear up the diagnosis. The leucocytosis seen in amebic dysentery is absent in infections with the strongyloides, and it is not likely that other chronic diarrheas and dysenteries will be confused with this disease, by those who keep the features already mentioned in mind.

TREATMENT.

The treatment consists in light diet, rest and tonics, with occasional heavy doses of thymol or Felix mass, followed by a purge. The milder cases are easily controlled by treatment, the most severe difficult to influence in any way. While the patient may be relieved of nearly all unpleasant symptoms, the complete eradication of the parasite is a matter of some difficulty. I know of no case, at present, where this has been accomplished. No medication will cause the parthenogenetic mother worm to appear in the stool, and until this occurs treatment can not be considered successful.

After the patient has been relieved of his more acute symptoms by ordinary measures of rest and diet, thymol may be given on an empty stomach, preferably in the morning; 10 to 12 grains given in capsules every five minutes for an hour, and following this, a saline, watching carefully for any symptoms of depression or collapse induced by the thymol; male fern may be used in place of thymol, given in a dram dose, followed one hour later by another dram. Between the periods of more active medication tincture of ferric chlorid may be given as a tonic. Gin has also been recommended.

I wish to recommend at this time, on purely theo-

retical grounds, the adoption of a more radical procedure in the treatment of this disease. I think we are justified in these cases in endeavoring to effect a radical cure at the cost of considerable patience, or even risk to the patient. It is true that we can easily control the majority of cases met with in this country by appropriate treatment, but, aside from the danger to life, there are other considerations which we have to meet. The disease imposes considerable disability on patients suffering from it, and it falls especially hard on those who earn their living by manual labor, as the frequent discharges interfere with any continuous employment. Again, in debilitated persons, especially the tuberculous, there is considerable indirect risk of life. Intestinal malassimilation is always a serious feature in this disease, and may result in developing a case previously latent, or in converting the chronic into the acute form. I would advocate treatment along several experimental lines, having for their object to render the habitat of the parasite unfavorable to development. The parasite is not especially robust, and can be destroyed external to the body by trifling untoward circumstances.

The presence of air, a moderate temperature and the power of free motion in a fluid or semi-fluid nutritive medium is essential to the life of the parasite. A moderate degree of heat is sufficient to destroy it. In the endeavor to influence the parasite unfavorably we can first proceed along the lines of depriving the parasite of air; this we should be able to accomplish, practically, for several days without any unsurpassable difficulty. The method by which we should accomplish this will probably be by means of a primary purge, followed by doses of opium sufficiently heavy to arrest intestinal peristalsis. Rectal alimentation should be used during this period to further guard against the entrance of air into the intestines. With the intestine empty and collapsed, and its walls in approximation, the small quantity of air remaining will soon be lost. In order to promote

the absorption of any residual air, recently heated wood charcoal may be administered with the opium. An endeavor to destroy the parasite by heat may be combined with this method. The administration of very hot fluids continuously by mouth may be combined with the preceding method. The intestine is very tolerant of hot liquids, and the temperature required is not great. In order to avoid the discomfort incident to swallowing very hot liquids, they may be administered by a stomach tube combined with some agent to facilitate the rapid passage of the fluids into the duodenum. These two methods may be combined in a procedure such as given below:

After a twenty-four hours' fast the patient is given two drams mag. sulph. in two pints of very hot water, one drop of croton oil is added to insure prompt passage from the stomach. The solution of mag. sulph. may be repeated again in an hour without the croton oil. After the patient has been thoroughly purged, opium is given by the mouth, combined with powdered charcoal, after which it is administered by rectum as long as may be considered necessary. Arresting of all movements for a week should be all that is necessary in the usual case. At the end of the stated period the solution of mag. sulph. is administered in the same manner.

PROGNOSIS.

The prognosis of the disease has already been discussed under the treatment.

I wish in conclusion to express my thanks to Dr. Thayer and Dr. Strong for the use of previous reports on this subject, both of whom I have freely quoted, and from the former of whom I have largely modeled my report. Also to Dr. Atkinson for valuable advice and assistance in the case.

DISCUSSION.

DR. CHARLES WARDELL STILES, Washington, D. C.—This parasite of Cochin China diarrhea must be more common in this country than is commonly supposed. It is not only found about

Baltimore, but four cases are reported from Washington and three cases from California. The life conditions necessary for this parasite are much the same as are necessary for the uncinaria. We can expect this nematode to correspond more or less generally to the geographic distribution of uncinaria.

With regard to the diagnosis, there are one or two pitfalls. We may find small worms and make the mistake of thinking that we have to deal with Cochin China diarrhea. Mistakes of this kind have been made by European and Asiatic physicians when they state that uncinaria runs through the same double cycle of development shown by strongyloides. Looss reported that he found in the feces rhabditiform embryos which were scarcely to be distinguished from the diarrhea worm. Various rhabditiform embryos are not uncommon in water and vinegar, but which have no connection with strongyloides, yet they may be found in the stools. If you wish to make a diagnosis of Cochin China diarrhea the simplest way is to find several rhabditiform worms in the feces; then a few of them should be raised to the free adult stage.

Another pitfall is likely to occur in the southeastern states. Occasionally the embryo of the American hookworm develops while the egg is still in the uterus of the female; and occasionally we may find in the feces of hookworm patients eggs with fully-developed rhabditiform embryos. If the egg shell is broken the rhabditiform embryos can then be distinguished from the strongyloides embryos only with difficulty and with the most careful examination.

Another pitfall is this: The pin worm (oxyuris) is viviparous, and here the embryos simulate the strongyloides embryos. In the tropics and in the Philippines strongyloides is a very common parasite, and we are likely to find cases of Cochin China diarrhea in almost any part of the country where we are dealing with soldiers who have served in the tropics. I do not know what treatment we are going to use. The results with thymol are not at all satisfactory.

Dr. Allen J. Smith, Galveston, Texas—This worm, I am satisfied, is a more common human parasite in the southern part of the United States than is at present realized. I have personally encountered examples of the embryo a number of times in the stools of patients from Galveston, but passed it by as a matter of interest rather than of importance, accepting in this idea the statements usually made in the text-books as to the rôle of this parasite in pathology. I must here (and I do so without hesitation) acknowledge that it was due to this mental attitude of mine that Dr. John T. Moore, Galveston, refrained from publishing an instance of the presence of the embryonic worms met in one of his patients a number of months ago, at a time when such publication would have ranked high

in order of priority of record of the parasite in this country. I had seen the worms before; had not recognized in the cases presenting them any definite clinical features referable to these zoöparasites; was aware that the original idea that they were the cause of Cochin China diarrhea had been abandoned and that writers generally were agreed that they were of little or no importance in pathogenesis. Under this impression I spoke of the matter lightly when Dr. Moore brought to the laboratory of pathology in the University of Texas his specimens for identification; because of which Dr. Moore refrained from reporting the case until some months later, when his interest was again aroused by Dr. Thayer's excellent discussion of the same parasite in the *Journal of Experimental Medicine*. Since Dr. Moore's case two others have been brought to my notice from the wards of Sealy Hospital.

On such reasons I would base my confidence that the parasite is probably frequent in the southern tier of states of this country. In the cases which I have known the discovery of the worms has been in a general way accidental, and there has been nothing to make them seem of actual clinical importance. In one of these cases, however, at the time of the examination, there was a decided diarrhea, in which possibly the strongyloides may have had some influence; but the parasites persisted after the subsidence of the diarrhea—and aside from this individual circumstance I have had no reason to think of the anguillula as of any pathogenic significance.

Dr. A. P. Ohlmacher, Gallipolis, Ohio—Some five months ago my interest in this subject was aroused by the discovery of *Strongyloides stercoralis* in an epileptic idiot, 16 years of age, one of the inmates of the Ohio Hospital for Epileptics. He was a native of Cleveland, Ohio, where he had lived at home until admitted to our institution, Jan. 7, 1902. There was no history of diarrhea before admission. His death occurred Nov. 23, 1902, from exhaustion. A diarrhea appeared three months previously, and three days before death the stools were blood-stained. He was confined in one of the closed wards. I examined the contents of the large intestine during the progress of the autopsy, finding the strongyloides; and subsequent search through the intestinal tract exhibited the worms from the pylorus to the anus. Both the adult female worm, eggs and developing embryos were found. My tentative diagnosis of strongyloides was confirmed by Dr. Chas. Wardell Stiles of Washington, who kindly examined some of the material from this case. This additional case, the first in the middle west of the United States, seems to emphasize the point made by Dr. Stiles and others in the present discussion—that the worm of Cochin China diarrhea is not a rare parasite in this country.

Dr. J. T. Moore, Galveston, Texas—My first case was dis-

covered in August, 1901, the patient appearing at the outdoor clinic of the John Sealy Hospital complaining of severe diarrhea, with cramps in the abdomen. He was rather nervous and slightly anemic. He was a mulatto. He had served in the Spanish war in Cuba. The stools were examined and the parasite of *Strongyloides intestinalis* was found. The specimen was submitted to Dr. Smith, who told me it was *Strongyloides intestinalis*. I remembered hearing of Dr. Thayer's case spoken of in Washington, I believe, at the American Congress of Physicians and Surgeons. Dr. Thayer kindly sent me a monograph of his cases. I reported my case to the medical society in June, 1902, and it is now in the hands of the American Medicine Publishing Company, the manuscript having been corrected. Recently I sent Dr. Price a synopsis of the case. I have now two other cases to be reported that I have since seen in the medical wards of Sealy Hospital. This parasite is, I believe, of common occurrence in Galveston and vicinity. I recently had another case under observation in which I saw both the rhabditiform and filariform parasites. I have never been able to secure the eggs of the strongyloides, although I have made cultures several times. I have seen many cases of uncinariasis.

CLINICAL DIAGNOSIS OF INTESTINAL PARASITES.

CHARLES WARDELL STILES, PH.D.
Chief of Division of Zoology, Hygienic Laboratory, U. S. Public Health and Marine-Hospital Service.
WASHINGTON, D. C.

In diagnosing intestinal parasites, we may use various methods, but the best method is undoubtedly that of a microscopic examination of the feces.

DIAGNOSIS BY SYMPTOMS.

The practitioner who relies on symptoms alone for his diagnosis will undoubtedly often successfully recognize the true nature of the trouble, especially in severe infections. For instance, when we are in the infected area of hookworm disease and meet a typical clay-eater we note a complex of symptoms which is quite characteristic, and in most cases of this kind a microscopic examination seems unnecessary. In numerous medium cases and in practically all light cases of this disease, however, a diagnosis on symptoms alone is uncertain unless we find severe cases, such as clay-eaters, in the same family. Outside of the infected area, probably few physicians would care to risk a definite diagnosis of this disease on symptoms alone even in the severe cases, while in medium and light cases it would be almost folly to do so.

BLOOD COUNTS.

Blood examination has become almost a matter of routine in the better hospitals and also in the private practice of many physicians, and in all such examina-

tions we should bear in mind the important fact that an increased eosinophilia is an indication of possible infection with animal parasites. Hence, when an increased eosinophilia is noticed, we should immediately make a microscopic examination of the feces for eggs or embryos of parasites, and if these are not found we should consider the possibility of trichinosis, especially in obscure typhoid-like cases.

GROSS EXAMINATION OF THE FECES.

Physicians in general appreciate the importance of a gross examination of the feces of infants, but less attention is given to an examination of the stools of adults.

In some cases a gross examination of the stool will reveal segments of a tapeworm, or in some cases of nematode infection the adult worms may be found. This, it seems, is more likely to occur after a patient has taken calomel or a small dose of some anthelmintic or after food has been withheld for twenty-four to forty-eight hours. In the first stage of trichinosis, especially where the diarrhea is severe, as in children, the adult trichinæ may sometimes be found. In hunting for these trichinæ the stools should be diluted with warm water and examined in a flat glass dish held over a black background.

The color of the stools is occasionally suggestive in case of infection with blood-sucking parasites, such as hookworms. The hookworm stool (such as usually seen in very severe cases) is reddish brown in color.

BLOTTING PAPER TEST.

In hookworm infections a very simple test may be made with blotting paper or even with an ordinary newspaper. Several ounces of the stool should be wrapped up in paper and allowed to stand for a few hours. In severe cases and in many median cases it will be noticed that a distinct reddish-brown stain is formed on the paper. In this test, however, there is an error of 30 per cent. or more.

Odor and Litmus Test.—While the odor of feces from parasitic infections is sometimes offensive, I have not been able to discover anything sufficiently characteristic about it to attract special attention. The litmus paper test is of no value, so far as my observations go.

MICROSCOPIC EXAMINATION OF FECES.

I believe that a routine microscopic examination of feces should be made in all hospital cases and also in all private cases so far as practical. The results which the Zoological Division of the Hygienic Laboratory has recently obtained in examining the patients at the Government Hospital for the Insane amply demonstrated the value of such work, for we found that 3 per cent. of the first 500 cases examined showed hookworm infection.

There seems to be an impression that fecal examination involves very disagreeable work. While it must be admitted that some stools are offensive, I would personally much prefer to make a feces examination than a sputum examination, for in working with feces one is much less likely to experience that nauseating sensation so commonly resulting from an examination of sputa.

TECHNIC.

The technic involved is exceedingly simple, since it is not necessary to stain the preparation. A portion of the feces, say 1 to 5 grams, is taken from the stool, wrapped up in a piece of paper and taken to the laboratory. Or, if it is desired to test for protozoa as well as for worms, it is better to place the specimen in a bottle so that the material will not dry. To avoid errors in interpretation it is well to make the examination as promptly as possible, although an experienced operator may examine the specimen even if it is a week or so old. In examining old specimens, however, it must be recalled that the older the specimen the more likely one is to fail to recognize protozoan infections.

An elaborate series of instruments may be used in preparing the slides if desired, or, if instruments are not at hand, a match will suffice.

For slides I prefer the large 2 by 3-inch, such as are used for large sections or for trichina inspection, rather than the 1 by 3-inch slide generally used in bacteriologic work. These larger slides are much more easily handled than are the smaller sizes. Having thoroughly cleaned the slide in order to remove any eggs which may have clung to it from some former examination, a drop of water is placed on it; next take a minute portion of feces on the end of a match or a glass rod and thoroughly mix it with the water so that the resulting mixture is not too thick; add more water if necessary. Next cover the mixture with a cover glass (square cover glass preferred) and the preparation is ready for examination. As many as two to four cover glasses may be conveniently placed on one slide and at least ten cover-glass preparations should be examined before a negative opinion is given.

In the work several very simple precautions are to be observed, namely, the specimen and the preparations are to be kept protected from flies, the hands are to be immediately cleaned if accidentally soiled with the feces, especially if nothing is known regarding the condition of the patient, and all glassware is to be thoroughly cleaned (hot water or hot water and soap). In case of the presence of *Tænia solium* in man, double precautions are to be observed, as the egg is infectious for man. In case of examining feces from dogs, the possibility of the presence of *Tænia echinococcus* and its transmissibility to man should be held in mind.

The biologic principle on which fecal examination rests is that the eggs of different animals are more or less characteristic. Accordingly, to properly carry out the examination one must become familiar with the eggs and embryos of different species, also with the form of certain plant cells and fibers which might be mistaken by the uninitiated for eggs or embryos of parasitic worms.

Not every egg found in the feces indicates an infec-

tion of the intestinal tract. In the case of infection with liverflukes, for instance, the eggs are discharged in the stools, and some of these forms resemble more or less closely the eggs of certain intestinal parasites. In lungfluke infection the eggs of *Paragonimus* may be found both in the sputum and in the feces. In bloodfluke infection the eggs of *Schistosoma* may be found both in the feces and in the urine. In certain cases, therefore, it is necessary to examine either the sputum or the urine in addition to the examination of the feces.

For illustrations of the eggs most commonly found, I refer you to any work on clinical diagnosis or to Bulletin 10, Hygienic Laboratory, U. S. Public Health and Marine-Hospital Service.

THE TREATMENT OF UNCINARIASIS.

THOMAS A. CLAYTOR, M.D.
Professor of Materia Medica and Therapeutics, Columbian University; Physician to Garfield and University Hospitals.
WASHINGTON, D. C.

The treatment of uncinariasis may be considered under three headings: First, the prophylactic treatment, or that directed toward the prevention of infection by the hook-worm; second, the means used to cause the expulsion of the parasite; third, the treatment of the anemia and debility resulting from the infection.

Prophylaxis is of very great importance, and is a subject which requires careful consideration. It seems probable that infection takes place chiefly through the mouth, either in infected water or food, or through contaminated hands, which carry the parasite to the mouth. Looss, however, has apparently proven that the larvæ may penetrate the skin. A possible entrance through the air passages has also been suggested, but does not seem probable.

A most important fact, and one which greatly simplifies the treatment of uncinariasis, is that there is no increase in the number of parasites in the intestinal tract of the victim. Each hook-worm is taken into the system in the larval state, and then develops into a mature parasite capable of producing eggs which must, however, be passed from the host before they can be hatched. Thus it seems probable that the removal of a sufferer from an infected locality to one free from infection would ultimately result in recovery, if the vitality had not been already too much taxed.

MODE OF INFECTION.

The eggs are deposited in the fecal evacuations, and are, under favorable circumstances, hatched in twenty-four to forty-eight hours. In the larval state they become scattered in water or soil, and are now ready to infect the host who swallows them, or whose skin they penetrate.

Since the primary infecting agents are to be found in the evacuations, the important question seems to be how best to insure their destruction before they become scattered in the surface soil.

Sandwith says "cleanliness alone is required," but this is not enough. Of course, washing the hands before eating would prevent a certain number of infections, but if we remember the possibilities of infection through the drinking water and in the use of raw vegetables, to say nothing of the theory which Looss has proven, of infection through the skin, it can readily be seen that washing the entire body several times a day would not suffice.

The first step, the all-important step, is toward the destruction of the eggs and larvæ. It is well known that the embryos can not live without air. In view of this fact, Sandwith[1] suggested that infected villages should be compelled to dig trench latrines outside the villages, to use the latrines alone, and to cover them daily with earth. Thus the larvæ, deprived of necessary air, would soon die and cease to be dangerous, and as a result the disease would in time be stamped out.

Sandwith would also insure the death of the embryos by exposing the latrines to the sun's heat, as they have been known to live in shaded earth contaminated by feces for more than six months. Giles (quoted by Sandwith) recommends ploughing the infected surroundings of villages, so as to bury the embryos.

Stiles[2] has advocated practically the same thing as has

1. Observations on 400 Cases of Ankylostomiasis, 1894.
2. Report on the Prevalence and Geographical Destruction of Hookworm Disease in the United States, 1903.

Sandwith for the localities in our southern states where uncinariasis is known to exist. He suggests the use of suitable water-tight receptacles for feces, which should be emptied frequently; that the people should be taught the necessity of making use of these receptacles at all times, instead of defecating at any convenient spot, where the eggs hatch and the larvæ become disseminated through the surface earth.

This education of the people can only be accomplished by the family physician. No matter what care is taken or efforts made to enforce such regulations, the stamping out of this serious disease is going to be a difficult and tedious problem, simply because its occurrence is almost exclusively among the most ignorant of our people. The discharges might be burned or disinfected by chemical agents, but these methods are not practical in the country districts where uncinariasis prevails. To me the use of trench latrines would seem to be the most practical method of disposing of the infected feces, as it would necessitate very little trouble and no expense, items which would naturally appeal strongly to the people.

THE MEANS USED TO CAUSE THE EXPULSION OF THE PARASITE.

As an anthelmintic in uncinariasis thymol has almost entirely displaced all other remedies. (The credit of discovering the cure of ankylostomiasis by thymol seems to be due to Bozzolo, in 1880.—Sandwith.) The most popular method of its administration is as follows: The patient is given a purgative the day before, and is placed on liquid diet. Thymol is then given in 15 to 30 grain doses (1 to 2 grams) for four doses, at intervals of two hours. If the bowels do not move spontaneously in twelve hours after the last dose, a purgative is given. If an alcoholic solution of thymol is used, the majority of the worms are passed in six to eight hours after the first dose. Therefore, the stools passed within this time are the most important to examine for the parasite. If

alcohol is not used they are seldom seen earlier than twelve hours after the first dose.

Sandwith has found that 4 grams (ʒi) given in two doses of 2 grams (30 grains) each at 8 and 10 a. m. respectively, are quite as efficient as the use of 6 or 8 grams given in three or four doses at intervals of two hours, and are less likely to cause the symptoms of collapse. He also omits the primary purge as unnecessary and conducive to exhaustion, and gives a dose of sulphate of magnesia or castor oil two hours after the second half of the thymol. It has been found best not to repeat the treatment more frequently than once a week unless there should be some specially urgent reason for a rapid expulsion of the parasites. This method and dosage, I think, will be found most satisfactory.

The fact that thymol is poisonous must not be lost sight of, and the patient should be carefully watched. Some authors have cautioned against the use of alcohol, ether, turpentine, chloroform, oil, glycerin and certain alkaline solutions during the administration of thymol because of its solubility in these media, thereby increasing the danger of rapid absorption and consequent poisoning. Other writers, however, advise the use of alcoholic stimulants, on the ground that the depressing effect of thymol is best combated by this remedy. No less an authority than Sandwith says: "Warned by the death of at least one of my patients immediately after digesting the thymol, I have always administered to feeble men 25 grams of brandy with each 2 grams of thymol, with the happiest results."

Stiles and Pfender in 1902 performed a number of experiments on dogs with thymol in various forms. From these experiments Stiles concludes by saying: "I am afraid of the use of alcoholics per os during treatment, and in the case of weak patients I should be more inclined to use a stimulant hypodermically than run the risk of dissolving the thymol too rapidly, or in too great quantity at one time."

Since thymol is the accepted anthelmintic in the treatment of uncinariasis, and as there seemed to be some difference of opinion as to the safety of its use in alcoholic solution, I instituted a short study of its action on dogs. I regret to say that time did not allow of as complete an investigation as I would have wished before the presentation of our conclusions before this Association. I hope, however, that a few facts of value have been determined.

The work has been carried on by E. E. Butterfield, a fourth-year medical student in the Columbian University and pathologic interne at Garfield Hospital, to whom I wish to express my thanks.

EXPERIMENTAL WORK WITH THYMOL.

Thymol was administered in powder, in alcoholic solution, rendered bland by the solution of olive oil, and hypodermically. Space does not allow of a discussion of previous observations upon the physiologic action of thymol; it suffices to say that our results did not agree in all respects with those of other investigators.

Thymol given in dry powder in small doses (2 grams) caused salivation, and later slight diarrhea. No marked or constant effect upon the temperature, pulse or general condition. Large doses (5 to 20 grams) cause profuse salivation, later vomiting, anorexia, disinclination to move unless aroused, diarrhea and slight elevation of temperature (0.5 F. to 1.0 F.), the latter appearing six hours or longer after the dose and lasting from twenty-four to forty-eight hours. Prolonged feeding in ascending doses of 2, 3, 4, 5, 6, 7, 8, 9, 11, 14 and 20 grams during four weeks caused no loss of weight, the weight of the dog on which this experiment was tried having risen from 22 to 26 pounds during the course of treatment. Eleven grams of the dry thymol is, apparently, not a dangerous dose to dogs of 22 pounds or more, if given carefully and after a meal. Four dogs were used in these experiments without a death.

Thymol Given in Alcoholic Solution.—Alarming

symptoms immediately following the administration of the alcoholic solution of thymol to dogs, seem to be almost always, if not invariably, due to aspiration of the irritating solution into the lungs. In seven dogs to which an alcoholic solution of from 2 to 10 grams was given an extensive consolidation of lung tissue was the only significant lesion.

If aspiration of the alcoholic solution of thymol occurs the symptoms are immediate. In a few seconds the dog staggers, seems unable to stand, and in a few minutes develops general convulsions with unconsciousness. The temperature falls 1 to 3 F., pulse and respiration become rapid, pupils are widely dilated, and respond sluggishly to light. Convulsions last from five to fifteen minutes after consciousness is regained, the temperature rises slowly, and in from six to twenty-four hours reaches a point 1 to 2 F. above the normal. This elevation persists for a number of days. A persistent cough develops, a fremitus may be felt over the lungs, and râles are heard. If recovery takes place the dog regains his appetite in one or two days, the cough gradually abates and the temperature falls to normal. This occurred in two out of seven cases. In some cases there is persistent cough, elevation of temperature, loss of appetite and emaciation which would probably end fatally if allowed to do so. Three out of seven presented these symptoms, but were killed on the third to the tenth day after dosage, extensive lung lesions being found at the autopsy. Of the remaining two, one died almost immediately, the bulk of the dose having entered the lungs, none being detected in the stomach (the stomach tube had accidentally entered the trachea). The other, though not very sick, was killed four hours later to ascertain the early changes in the gastrointestinal tract.

Of the seven dogs experimented on, two recovered, and at the end of three weeks were apparently well, save for a slight cough; three would probably have died; one died; the remaining dog would undoubtedly have recovered, as the lung lesions were not extreme.

In conclusion it may be said that doses of 2 grams or more in alcoholic solution are unsafe for dogs, owing to a tendency to aspiration pneumonia.

Thymol in Alcoholic Solution Rendered Bland by the Addition of Olive Oil.—To avoid the irritating effect of the alcoholic solution, the drug was dissolved in alcohol, and enough oil added to render the mixture non-irritating, as tested by taste. Four grams were then given successfully to dogs of 16 to 17 pounds, without depression or fall of temperature. Six grams were given to a 17-pound dog, with a resulting fall of 2 degrees of temperature four hours after the dose was given, and lasting for six days, with depression, anorexia, diarrhea with bloody stools, emaciation (8 pounds lost) and coldness of the surface and extremities.

The autopsy, performed six days after the dosage, showed extensive subacute gastrointestinal inflammation with only two pea-sized areas of congestion at the base of the left lung.

Conclusions: Doses of 2 to 4 grams of thymol in alcoholic solution rendered bland with oil are safe for dogs of 17 pounds or more.

Thymol Hypodermically.—One 16-pound dog was given an alcoholic solution hypodermically to determine whether any toxic symptoms would develop from this manner of administration.

Ten c. gm., 0.20 gm., 1.00 gm. and 1.00 gm. repeated were given at intervals of one or two days without any apparent effect, except inflammatory swelling at the site of injection. Autopsy was performed one week after the last dosage. No pathologic lesions were found, except congestion of the lungs, which may have been produced during the process of anesthetization, which, in this case, was attended with considerable difficulty.

Autopsies on Eight Dogs.—Microscopic examination of the viscera of eight of the dogs to which thymol had been administered showed no changes which were not attributable to the local irritative effect of the drug. The

liver, spleen, kidneys and heart muscle were normal. The changes in the lungs varied from intra-alveolar hemorrhage to bronchopneumonia in cases where aspiration had occurred. The condition of the stomach and intestines varied from mild congestion to marked inflammation, most intense after the administration of the drug in alcohol and oil; least intense after the dry. The probable reason why the inflammation was not so marked after the administration of the simple alcoholic solution was that it was so irritating that much of it was drawn into the lungs by the violent coughing it caused. In no instance were albumin or casts found in the urine at autopsy.

CONCLUSIONS.

1. The alcoholic solution of thymol is dangerous to dogs, even in small doses, chiefly because of its tendency to produce inflammation of the lungs by aspiration.

2. The alcoholic solution, even when rendered bland by the addition of oil, is dangerous in doses of more than 4 grams to dogs of 16 or 17 pounds, because of its irritative effect upon the gastrointestinal mucous membrane.

3. In dry powder the drug may be given to dogs in large doses without serious results, no depression, no fall of temperature.

4. Gram doses given hypodermically cause only local inflammation.

5. There is probably no danger in giving 2 to 4 grams, followed by 1 to 2 tablespoonfuls of brandy, to an adult human being, if followed in a few hours by a purgative such as castor oil or magnesium sulphate.

6. It is probable that the alcoholic solution obtained in this way would be more efficacious against the parasites, as seems proven by their much earlier appearance in the stools after the use of the alcoholic solution.

THE NUMBER OF DOSES REQUIRED TO EFFECT A CURE.

As to the number of doses of thymol which are usually required to effect a cure, I can not do better than to again

quote Sandwith, who says: "In 184 patients I was able to prove the absence of worms by giving at least two, and sometimes more, doses of thymol after the last appearance of the parasite in the stools. The average number of doses required proves to be 2.6. Forty-two men were cured after only one dose, fifty-eight after two, forty-two after three, twenty-five after four, nine after five, and four after six doses of thymol. Two cases required seven attacks, and two others eight."

In my own case, after giving thymol at least nine times in a period of nine weeks in two doses of 2 grams each, separated by two hours, and followed by a purgative, there were found at the autopsy twenty-nine worms in the small intestine. This serves as a warning that in some cases the parasites are extremely difficult to exterminate, and require a prolonged course of treatment. The stools should be examined weekly for eggs, and as long as they are present the thymol treatment should be continued.

As to the resulting anemia and debility, a general tonic treatment with iron and abundance of good food and fresh air are indicated.

DISCUSSION

ON PAPERS OF DRS. STILES, SMITH* AND CLAYTOR.

DR. W. G. HARRISON, Talladega, Ala.—After hearing Dr. Osler speak on this subject in his wards I felt sure that I saw some cases that were affected with hook-worm disease. When I came home a physician called attention to some suspicious cases at the Deaf, Dumb and Blind School. Several cases were found at the time the state medical association met with us, when Dr. Stiles was our guest. He confirmed the diagnosis in one of the four cases. A microscopic study of the case was not made until the early part of the last week. It was my purpose to treat the case with thymol, but at the suggestion of Dr. Stiles we gave them one or two remedies before resorting to thymol. These children were all in the schools. The youngest was 7 and the oldest only 18 years old. All were extremely anemic. No blood counts were made. Hemoglobin in the lowest was 25 per cent., in the highest 43 per cent. We were careful to make an accurate temperature record. The

*The paper of Dr. Smith has not been furnished for publication.

temperature of each of these patients was not much elevated. In four it was over 100 F., in three between 99.7 and 100 F. In only one was I able to demonstrate the malarial organism, so they could not have been due to latent malaria. In all cases there was a heart murmur, in three a systolic, in four an aortic. In four out of seven cases there was a marked pulsation of the vessels of the neck. It is rather an interesting point that Dr. Stiles himself recognized the disease in whites, but said he was not sufficiently familiar with its characteristics in the negro to recognize it. It was stated that it was likely one of the pupils would be affected with the hook-worm, and many students were examined and hook-worm disease found. Subsequently, in one of the schools, being unable to pick out the likely subjects, I asked the superintendent if there were any who appeared to be especially lazy, or any pupils who would not play, who would not take exercise, or frolic, or run or jump. He answered: "Yes, I have two boys who are so lazy that I can not get them to do anything." I examined those two and found the eggs in both. Three out of the seven cases I examined were among the blind. I saw two cases among the mutes. The peculiarity about the eyes was noticed. I had heard Dr. Welsh speak of the "stare." As soon as we were inside I said: "Doctor, I guess you will find the stare." Subsequently we went through the carpet factory and found in three out of seven cases the stare.

The treatment of these seven cases was as follows: Two were given four grains of calomel every three hours for four doses, and after a couple of days Epsom salts were administered. Twenty-four hours later the worms could be found in the stools. Then, for each patient, for two days, thymol was given in 15-grain doses at intervals of three hours. On the following morning there were found in the stools from 40 to 50 worms. The above was the treatment given in two cases. The third case was given 10 grains of salol every four hours for four days. No worms were found in 24 hours. In two days 10 grains of thymol were administered, and this patient passed 28 worms during the next 24 hours. In case four calomel, grains four, and santonin, grain one, were given every three hours until four doses were given. No worm inside of 24 hours. The next day two doses of 15 grains of thymol were given, and then there were passed between 60 and 70 worms. Case five fasted two days by accident, and this patient passed two worms before any medicine was given. That patient was given 10 grains of salol and 4 grains of calomel and 1 grain of santonin every three hours until four doses were given. This patient then passed 10 or 12 worms. On the succeeding day this patient was given 3 grains of thymol every three hours until three doses were taken, and on the following morning there was found in the stool 27

worms. Case six was given 15 grains of thymol every three hours for two days, and this patient passed a large number of worms, over 250, and probably four times as many more. Case seven had an incomplete record. He passed many worms.

Dr. E. D. Bondurant, Mobile, Ala.—My attention was first directed to the subject under discussion by a fellow practitioner in Mobile, Dr. W. T. Henderson, who told me of three cases which he had seen, and which from the peculiar edema shown he believed to be uncinariasis. The feces from those cases were submitted to me for microscopic examination, and in all of them I found the ova of uncinaria. Since then I have succeeded in interesting a number of my students and other doctors in the study of the disease, and have had a large number of fecal specimens sent to me for diagnostic examination. In this way I have already diagnosed more than fifty cases of uncinariasis in Alabama, chiefly from the counties of Mobile, Monroe, Escambia, Covington and Crenshaw, the greatest prevalence seeming to be in the sandy pine hill country west of the city of Mobile. In south Alabama there can be no question that the disease is more common on the sandy land than in the black lands or on dry formations. Uncinariasis is surely a very common complaint in our part of the world. It is rather a significant fact that in every instance in which uncinariasis has been suspected and fecal matter submitted for miscroscopic examination, I have been able to demonstrate the presence of hook-worm ova.

Being a neurologic specialist I have not had very great opportunity of studying the symptomatology and treatment of this disease, but during the past four months I have had to deal with uncinariasis as a complication or possibly causative factor in sundry cases of nervous disease. These patients, six or seven in all, have shown the anemia, listlessness and general mental and bodily enfeeblement already dwelt on by the authors of the papers under consideration, and all of them have been treated with thymol. The usual dose has been 60 grains, given in six capsules within two hours' time, preceded by calomel and followed by a saline. The results have been favorable without exception, numbers of worms being expelled and subsequent rapid improvement shown in the general condition of the patient.

Attention should, however, be called to the possible dangers of these large doses of thymol. One of my former pupils wrote me that he gave an 8-year-old boy 60 grains and came near killing him, the child passing into a conditon of coma with spasmodic movements and seeming at the point of death for hours; he was cured of uncinariasis, however.

Regarding the number of worms present, I know of one case in which a patient passed 953, and another instance, reported

to me by Dr. Parker of Crenshaw County, in which "over 1,700 worms were counted, and there were more beside."

I have made careful examination of the blood in four cases, and in each one found a great diminution in hemoglobin percentage, with comparatively slight reduction in the number of red blood cells.

Now that Dr. Stiles has overwhelmed us with evidence and shown us how anybody can diagnose uncinariasis, we should not forget that the disease was recognized by Dr. Allen J. Smith ten years ago, when no one knew much about it, and its frequency was unsuspected.

Dr. C. A. Mohr, Mobile, Ala.—Unless one has had a case of uncinariasis one can not appreciate the great work accomplished by Smith and Stiles. We in Alabama can fully appreciate it, because they have been so plentiful there. For a long time we were unacquainted with the disease and were treating for this, that and the other thing, and especially for malarial anemia. In fact, the poor little devils have taken iron enough to build battleships, and without any effect on the anemia whatever. Only when our attention was drawn to the true nature of the trouble did we get any good results. The first case in Mobile was recognized by Dr. Henderson, and he made a diagnosis from the clinical symptoms only. The pulsations in the carotids, the intense anemia, the lowered amount of hemoglobin, the systolic murmur, etc., are all such striking symptoms. How the anemia is accounted for I am at a loss to say; I do not know whether it is due to the loss of blood, or whether it is produced by the toxemia, or possibly the secretions of the worms themselves. It is hard for me to believe from the amount of the anemia which is so intense as in chlorosis and the loss of hemoglobin, etc., that it is due to anything so far attempted in the way of an explanation. But I am sure that many cases of so-called "idiopathic anemia" will be found to be due to some parasitic disease.

Dr. G. W. McCaskey, Fort Wayne, Ind.—My experience is limited to but three cases seen in northern Indiana, occurring over one year ago. Two cases were in Syrians, and therefore imported, while the other was an American.

I wish to emphasize the importance of a routine examination of the feces, not only in hospital cases, as advocated by Dr. Stiles, but in private practice as well. For several years I have been in the habit of making this routine examination of feces in practically all cases. A number of writers think that it would not be necessary to do this; Ewald, Einhorn and others think it is not necessary. In 1895 I read a paper advocating this routine examination of the feces, and I have been keeping up such examinations ever since. It is not especially unpleasant; less so than sputum examination. My principal object in getting up to-day is to plead for this

routine examination of the feces in all cases of obscure chronic nutritional diseases, in which, among many other things, important parasitic ova may be found at any time.

Dr. Claude A. Smith, Atlanta, Ga.—This disease is not especially confined to the sand belt; it is also found in the mountain regions. Through observations that have been carried on in Atlanta and adjacent territory, and especially by examination of students in the Atlanta College of Physicians and Surgeons and the Southern Dental College, I have traced the disease from the Carolinas to Texas.

Almost every country practitioner in the South sees every year any number of cases of anemia, and many of them of the profound type, which are often classed as malarial cachexia, and they have been treating these cases with quinin, iron, arsenic, etc., with only slight temporary improvement, and I have found many such to be cases of uncinariasis, and the treatment with thymol has given positive results, which were sometimes remarkable.

Since we have recognized in the South that this disease is so very common, the point which concerns us most at the present time is by what means does it reach the intestines. During my early observations I was inclined to the common belief that it is taken into the body in the drinking water, but later observations led me to the conclusion that the larvæ gain entrance through the skin of the feet, as first pointed out by Bently, producing the ordinary "ground-itch," which is so common in the South in the summer after a period of rainy weather. In fact, I have found that it is possible to locate cases of uncinariasis that you would not otherwise suspect by simply getting the history of the patient in regard to attacks of ground-itch, and I have found that every person who has had an attack of ground-itch within a period of seven or eight years has the uncinaria in his intestines.

My first experience with this disease was in December, 1901, when, on making a postmortem examination on a case which had died at the Grady Hospital in Atlanta, I found over 300 of the uncinaria in the small intestine, and on looking up the history of the patient I found that he was from Florida and had never been out of the South. As this case was evidently contracted in the South I naturally concluded that it must be endemic, and through the kindness of Dr. S. H. Green I obtained the stools of twenty convicts who work in the damp clay in one of the large brickyards near Atlanta, and of these twenty I found two cases of uncinariasis. These cases were reported by me at the session of the American Medical Association at Saratoga last year in the Section on Pathology and Physiology. Since that time I have found the disease to be so common as not to be practicable to keep a record of all cases.

The reason the full extent of the disease was not recognized

sooner was because it is a disease of the country and not of the city, and it abounds under unsanitary conditions.

In regard to the systematic examination of the stools of all patients, as mentioned by Dr. Stiles, since the first case appeared at the Grady Hospital I have made a systematic examination of the stools of all patients, and during this time have found only three cases in the hospital. The rules of the hospital require that all applicants for admission must reside in the city, and this accounts for the small number. As I have mentioned before, you can locate the cases with a fair degree of accuracy by securing a history of the patient with reference to attacks of ground-itch within seven or eight years.

Dr. James B. Herrick, Chicago—I wish to call attention to the fact that although this is a disease of the tropics, we in the North should keep our eyes open for imported cases. This last year Dr. Capps of Chicago observed and reported a case that came from Panama, and who reached Chicago after a prolonged stay in the hospital in New Orleans. In this case it is interesting to note the occurrence of 13 per cent. eosinophilia with very marked anemia, the large number of eosinophiles calling attention to the possible existence of an intestinal animal parasite.

Dr. Wm. Britt Burns, Memphis, Tenn.—I would like to refer to two cases which did not show uncinaria. The first case was an engineer who had been in a logging camp, sometimes having occasion to drive teams through lakes of mud. I had examined him a number of times for the malarial parasite, which I found, and thought he had a malarial cachexia. The man came to me in Memphis three months ago. He had fainted and I restored him in my office. He was then placed in the hospital. He had a very marked anemia. He did not remain long enough for me to make a complete blood count. The blood was found to contain the malarial parasite. He had no temperature at that time. The stools did not show uncinaria or other intestinal parasite. This patient had marked symptoms of sleepiness; he slept every time he sat down. He was an engineer at that time and when it was time for him to pull the cord attached to the whistle, on one occasion he raised his hand to pull the cord and went to sleep, and passed the station. He had some diarrhea alternating with constipation, and his bowels would gurgle, as he expressed it. He had some colic. The stools were white. I wished to get him for further examination, but could not.

The second case was a soldier from the Philippines who came to me with diarrhea, and he said that he had had it for some time, possibly a year. He had been in the hospital for six months. Coming on to the pathologic service six weeks ago I made several examinations of his stools. There were

no malarial parasites found in the blood. The parasites that I found in the stools were of this character: (Blackboard demonstration.)

I would like to know more regarding the character of the stools in these cases of uncinariasis and of other parasitic diseases of the bowels. I would also like to know the condition of the spleen and liver, the skin and sclera, and also something of the pathology of the mucous lining of the gut, whether there is ulceration or much involvement.

DR. CHARLES WARDELL STILES, Washington, D. C.—With regard to ground-itch, I may say that this is exceedingly common in people affected with uncinariasis; but so far as I understand at present it is also found in people who have no hook-worm disease. I am informed by physicians of Georgia and Florida that practically every inhabitant of these states, except persons living in cities, have had ground-itch at one time or another. Undoubtedly uncinariasis infection may take place through the skin, as has been demonstrated experimentally by Looss of Cairo, Egypt. As to infection through the drinking water, I think this is possible, but the chances are that such method of infection is not so common as might be supposed, for the reason that the larvæ of hook-worms sink in water. Infection through cotton, as was suggested in connection with one case, I consider practically excluded on account of the dryness of the cotton.

Length of life of parasite: I have two cases where the parasites have lived six years, and one case six years and seven months, after the patients entered an orphan asylum.

Disinfection: I consider chemical disinfection of premises impractical. Yards may, however, be disinfected with fire, either by spraying burning oil or by scattering straw around and setting it on fire.

Number of parasites present: Dr. Harrison's case, which passed over 1,700 worms, is interesting, and so far as I know now holds the world's record. Ashford sent me about 1,400 worms from one case in Porto Rico.

Classification of the parasite: Dr. Smith has spoken of the validity of the American species. My studies on the anatomy of hook-worms have been interrupted, but it is now easy to foresee that the old genus *Uncinaria* will have to be subdivided. Worms like *Anchylostoma duodenale* and *A. caninum* form a distinct group characterized by the presence of recurred hooks in the mouth; these parasites should be classified in the genus *Anchylostoma*. The worms without buccal hooks, but provided with buccal lips, belong at present in the genus *Uncinaria*, but these forms may be divided into several subgenera, at least, which may eventually be given the rank of genera. The typical subgenus is *Uncinaria*, with *U. vulpis* as a type. Species with non-symmetrical bursa and with

prominent buccal tooth, like *Uncinaria radiata* of cattle and *U. trigonocephala* of sheep, may be placed in Railliet's (1902) *Bunostomum*. *Monodontus* (Molin), 1861, should be based on *M. semicircularis*, and apparently presents, according to Molin, a symmetrical bursa with dorso-median ray of the usual type. *Uncinaria americana* seems to take a very isolated position, with its buccal capsule more or less similar to that of *Bunostomum*, but with a symmetrical bursa in which the dorso-median ray is of a very aberrant type. I believe we are justified in recognizing for this species a distinct subgenus (for which I would suggest the name *Necator*—a murderer), which may eventually be raised to generic rank. *Uncinara lucasi* is still different in certain characters, notably of the anterior end, and will probably call for the erection of a new subgenus. *Agriostomum* and *Characostomum* (Railliet), 1902, will probably stand as distinct genera.

The anemia: I agree with the view that it is difficult to explain the severe anemia and the nervous symptoms by the mere extraction of blood. In all probability the cervical glands of the parasites produce a poison which could be readily absorbed by the wounded mucosa of the intestine.

Diagnosis: As to the unpleasantness of a fecal examination, I frankly admit that I would much rather examine the feces than the sputum of a tuberculous patient.

Topography: I do not recall ever having stated that hook-worm disease is confined to sandy soils. What I claim is, that it is pre-eminently a disease of sandy regions.

Color of stools: The color of hook-worm stools is frequently a peculiar reddish brown, a quite different shade from anything I recall in connection with feces I have examined in Washington. Still, this color is not constant even in very severe infections. Nor is the blotting-paper test constant; in fact, it should never be relied on to the exclusion of the microscopic examination, for the chance of error is too great. I suggest the test simply as a convenient confirmatory test in severe cases for physicians in the infected district who are unable to make the microscopic examination. Blood in the stools is by no means constant. It does, however, occur.

Dr. Allen J. Smith, Galveston, Texas—It should be kept in mind, in comparing the group of cases which I have presented with the types of cases which have been mentioned in this discussion, that these instances represented a mild form, presented but few symptoms and were found in the course of a routine examination of a large group of persons undertaken because in some one or more of these persons it was certain the parasite existed. In the first and second of these cases the presence of an eosinophilia, found when searching for the malarial hematozoön, suggested the investigation of the stools for the uncinaria known to be present somewhere in the gen-

eral group of students; and naturally the success of the search in these two early cases led to the thought of the value of the symptom as at least of suggestive force. In those cases of which I have records of the differential leucocyte count the proportion of eosinophiles ranges from 6 to 23 per cent.

There can be little if any doubt of the fact that one mode of acquirement involves the penetration by the embryonic worms in dirty water or in the mud adhering to dirty objects through the skin and tissues to their ultimate place of abode in the alimentary canal; while it is quite probable, too, that in many cases the entrance to the intestine is direct from drinking unclean water or eating dirty vegetables moist with damp earth. I do not feel that Dr. Stiles' view of the improbability of their transference by cistern water has included conditions which commonly prevail in the frequent over-ground cisterns found in many southern localities. There is easy chance for entrance of the ova to such cisterns by their being blown with sand and dust to the house-tops, whence they may be washed by succeeding rains into the cistern. Sinking to the bottom of such receptacle they usually find what seems to me to be a not unsuitable surrounding for their incubation, providing they have retained their vitality, in the layer of sand or mud, mixed as it often is with charcoal commonly introduced into cisterns with a view of purification of the water; and as the faucets of these cisterns are placed close to the bottom it seems possible that the embryonic worms may be drained with the water used for drinking purposes or otherwise.

I also wish to lay some stress on the relationship which I believe may exist between this parasite and malaria. In six of the eight cases which I have mentioned there is a history of malarial complication, temporarily influenced by quinin, but invariably recurring after longer or shorter intervals. In several of these cases the demonstration of the malarial organism was unquestionable, but invariably was made only after repeated search and with more or less difficulty. While these malarial outbreaks were temporarily restrained by the administration of quinin, during the period of prevalence of the hook-worm in the intestine the malarial recurrences persisted indefinitely, the use of the quinin producing no permanent effect. All this ceased after the parasites were removed from the intestine, subsequent administration of quinin being promptly and permanently followed by the disappearance of the malarial symptoms. It might be suggested that in an indirect way the intestinal parasites have an influence in bringing about a persistence of the malarial infection—probably by lowering the natural powers of the system of the host in reaction and antagonism to some small residuum of the malarial infection after partial destruction by quinin, so that subsequently that residuum more surely may increase to a

degree capable of producing the recurrence noted in such cases. With the removal of the worms there would seem to return the natural resistive influence of the body, and this joined with the potency of the quinin leads to complete and permanent destruction of the malarial infection. Doubtless there are other explanations possible for what in my experience is a very real relation of the character mentioned; but the idea suggested does not seem improbable. In such cases of an apparently malarial infection, with few organisms ordinarily recognizable, and these often missed, and with an irregularly recurrent type persisting after the use of quinin (although the latter may have been temporarily beneficial), I would urge that investigation be made for a possible complication by uncinariasis. If the latter be recognized I am confident that the experience I have mentioned may be repeated, and that by first removing the intestinal parasites it will be found that a difficult and persistent malaria will become readily amenable to the ordinary administration of quinin. It adds but little difficulty to the labor of diagnosis. The hook-worm egg is one of the most easily detected of microscopic objects in the fecal material; there is no complicated technic required in the examination of the material, and probably five minutes' attention as an average will suffice for the added work.

The relation between hook-worm diseases in man and in the dog is an item in which I am satisfied that I have publicly expressed mistaken ideas. In the discussion of this matter in 1901 before the Texas State Medical Association, I commented on the similarity of our human uncinaria and the dog hook-worm, and I expressed some question as to a possibility of identity as well as a possibility of the dog being an agent in scattering the parasites where man might acquire them. From the work of others as well as from the clear differences I have since learned to recognize between these parasites I fully realize the mistake into which my ignorance then led me.

I am satisfied, too, of another mistaken suggestion I made at the same time, namely, of the possibility of incubation of the eggs in the intestine of an original host, and thus a chance of permitting a slight infection to increase to a serious degree and to make itself permanent. While I am unable as yet to entirely set this idea aside as an occasional possibility, there is no doubt in my mind that it is essentially always untrue. That it is not impossible I would infer from the fact that occasionally the ova in very fresh feces are found to contain well-formed embryonic worms; and that rarely even in the ova within a gravid female worm an embryo may be found. Of course, too, the segmentation seen in the ova ordinarily is evidence of some procedure of incubation; and should the egg happen to be retained unduly in some groove or in a mass of mucus adherent to the mucous membrane of

the intestine, it might on rare occasions develop into the freed embryonic worm. It is impossible for me to indicate why this does not ordinarily take place. I have noted in the incubation of ova in the fecal material that the process has proceeded more quickly and certainly at the ordinary room temperature than in the incubator at body temperature; and although there are probably other factors than mere temperature at work in this connection, it may be one element in restraining incubation in the intestine.

DR. THOMAS A. CLAYTOR, Washington, D. C.—Regarding the elevation of temperature in these cases, it has been claimed that fever is unusual. I think, however, that this depends on the degree of anemia. In intense anemia, no matter from what cause, moderate fever of an irregular type is frequently seen.

Several of the gentlemen have spoken of symptoms by which they could diagnose uncinariasis—such as paleness of the skin, hemic murmurs, pulsation of the vessels of the neck, etc., which mean nothing but anemia, and hence are only suggestive. Without examination of the stools I do not believe a positive diagnosis can be made. It is an easy matter today to have such examinations made.

Regarding the dose of thymol to be administered, it is from 30 to 60 grains (2 to 4 grams). Sandwith, who has had a very large experience in the use of this drug, gives 30 grains at 8 o'clock, 30 at 10 and after two hours a purgative. In weak individuals he gives an ounce of brandy for every 30 grains of thymol.

It has been said that the American hook-worm is more readily gotten rid of than the old-world variety, because of the absence of hooklets in the former. In a case which I have reported two months' treatment with thymol failed to entirely free the sufferer from the parasites, though they were of the American variety.

The weekly treatment with thymol should be continued until the eggs disappear from the evacuations.

BACILLARY DYSENTERY (SHIGA).

CHAS. F. MASON, M.D.
MAJOR AND SURGEON U. S. ARMY.
FORT SAM HOUSTON, TEXAS.

GENERAL REMARKS.

Dysentery has been, from the earliest times, a disease of great importance, and especially has this been the case in the tropics and in the camps of great armies in the field. My own impression, gained from something over two years' service in the tropics, is that intestinal disorders far outnumber all other diseases, and that of these, dysentery is of overshadowing importance, both on account of its high rate of mortality, and because of the invaliding which results from it.

In the medical history of the War of the Rebellion something over 259,000 cases of acute dysentery and 28,000 cases of the chronic type are reported to have occurred in the Federal Army alone; and, again, during the Spanish-American War the same disease played havoc among our troops in Cuba, Porto Rico and the Philippines; in the South African campaign it was also widely prevalent.

In the tropics it is endemic at all times, and its importance may be gathered from the observations of Strong that of 1,830 cases in soldiers admitted to one hospital in Manila in sixteen months, only one-third were returned from there to military duty, and that the deaths from dysentery in the Army in the Philippines were nearly four times as great as from any other single disease.

For many years an epidemic form of bacillary dysentery has prevailed in Japan in the late summer and fall; in 1899, there were some 130,000 cases, with more than 26,000 deaths.

The disease occurs all over the world, increasing in frequency and importance the nearer we approach the tropics. In the temperate zone it occurs sporadically, at times assuming an epidemic form, which, even in cold climates, may be widespread and fatal; and this is especially apt to be the case in institutions such as insane asylums, or other places where there is overcrowding and bad hygienic conditions. Dysentery is especially a disease of the late summer and autumn, and in the tropics is most prevalent toward the end of the rainy season, though it occurs at all seasons. Age and sex have apparently no other influence than that resulting from the greater exposure incident to the life of the male adult.

ETIOLOGY.

The dysenteries have been variously classified on epidemiologic, clinical and pathologic-anatomic grounds, and the varieties described by different authorities have been as numerous as their points of view.

On the one hand, some writers believe in the essential unity of all the dysenteries, while on the other it has been held that many forms of bacteria and several varieties of amebæ were each responsible for a particular form of the disease. Since the discovery by Losch, in 1875, of amebæ in the stools of dysenteric patients, these organisms have been regarded as the cause of tropical dysentery by many authorities. Between that date and 1897 nearly all the varieties of intestinal flora have been accused of being the causative agents of dysentery; but it was not until the latter year that Shiga, studying an epidemic of acute dysentery in Japan, was able to demonstrate the constant presence, in the intestinal discharges and in the inflamed mucous membranes, of

bacilli which were not found in the intestinal tract of healthy subjects or in those suffering from other diseases, and using the agglutination test of Widal, announced the year before for typhoid fever, proved that the blood serum from his dysenteric cases gave the specific agglutination with the newly-isolated bacillus.

Flexner, in 1900, found the same bacillus in dysenteric stools in Manila, but was not satisfied that it was a specific organism. Later in the same year Strong and Musgrave confirmed his findings, and expressed their belief in the specific character of the organism. Since then the same bacillus has been isolated from dysenteric stools by Krause in Germany, Flexner and Duval and Vedder in the United States, Deycke in Turkey, Frosch v. Drigalski and Conradi in Prussia in the dysentery supposed to have been brought from China during the recent war, and by Foulerton among the English troops from South Africa. Still more recently Duval and Bassett have isolated the same bacillus from the stools in the summer diarrhea of infants.

It thus seems to be established that there are, etiologically, at least, two distinct types of dysentery, one ordinarily acute and due to the *Bacillus dysenteriæ* of Shiga, and the other usually chronic without the Shiga bacillus in the stools, and due to the ameba dysenteriæ. To the bacillary types belong the epidemic dysenteries, and the sporadic dysenteries of temperate climates, as well as the summer diarrheas of infancy.

Bacillary dysentery, then, is an acute infectious disease due to the *Bacillus dysenteriæ*. It is characterized clinically by frequent loose passages containing blood and mucus, by pain in the abdomen, tenesmus, and more or less fever.

Anatomically, there is catarrhal inflammation, fibrinous exudation, coagulation necrosis, and finally, ulceration. The inflammation affects chiefly the large intestine, but sometimes the lower end of the ileum is involved as well.

THE BACILLUS DYSENTERIÆ.

These bacilli are small rods with rounded ends, resembling the typhoid and colon organisms. They are slightly motile, flagellated, stain with ordinary dyes, decolorize with Gram, and do not liquefy gelatin. Their resisting power is slight, being readily killed by weak antiseptic solutions, and by an exposure of half an hour to sunlight.

The bacillus is not readily found in the evacuations for the first few days nor in the subacute and chronic stages, but by the end of the first week it is found abundantly in the blood and mucus discharges. Toward the end of the attack it disappears to reappear in relapses.

In the intestines it is found in the fresh exudate and in the deeper layers of the ulcers. It is also found in the mesenteric glands, but never elsewhere.

The blood of dysenteric patients agglutinates the *Bacillus dysenteriæ* in dilutions of 1 to 20 to 1 to 130, depending on the severity of the case and the stage of the disease. According to Shiga, this alone is not sufficient evidence, and to be positive the agglutination should progressively increase in intensity. Agglutination appears, according to Strong, as early as the third day, and is usually marked by the fifth or sixth day; but, according to Shiga, it is not positive until the second or third week, and is not therefore, of great importance in diagnosis. It has been found as late as eight months after recovery.

The value of the bacillary diagnosis is lessened by the fact that the bacilli can not often be found in the early stages or in the mild cases, but when the morbid process is low down in the colon or in the rectum, they can generally be found without difficulty. Agar plates are made in the usual manner, and after twenty-four hours' incubation the colonies are examined, and those which are least developed and most transparent are tested as follows:

1. Agglutinating capability with immune serum.
2. Cultivation upon glucose-agar.
3. Cultivation in milk.

If the agglutination reaction is positive, no gas is produced, and milk is not coagulated, the microorganism is *B. dysenteriæ.*

Shiga gives the following proofs that his bacillus is the specific cause of dysentery:

1 It is present in all cases.
2. It is never found in other diseases or in healthy persons.
3. Its presence in the stools corresponds in degree with the intensity of the morbid process.
4. It is found in the lesions in the intestinal wall.
5. It, or its toxins, cause intestinal hemorrhage.
6. It is agglutinated only by the blood of dysenteric patients.
7. The agglutinating power of the blood increases in intensity with convalescence.
8. Pfeiffer's reaction is marked.
9. Immune serum is of therapeutic and prophylactic value.
10. The infection of man: this occurred in one case where a pure culture was intentionally given to a condemned criminal, and in another where a culture was accidentally drawn into the mouth. There have also been at least two other cases of accidental laboratory infection.

PATHOLOGIC ANATOMY.

The lesions are those of a catarrhal, diphtheritic and sometimes hemorrhagic inflammation of a part or the whole of the large intestine, and occasionally of the lower portion of the small intestine.

The inflammation is most marked opposite the flexures of the colon because of the tendency of fecal masses to lodge there and set up inflammation through irritation. The changes begin in the mucosa and extend to the deeper parts, in contradistinction to those of the amebic type, where they are most marked in the submucosa. There is extensive coagulation necrosis; but, according to Strong, few or no real ulcers. Shiga, in Japan, reports the colon as riddled with small, shallow, clean-cut ulcers. The anatomic picture, however, is very different from that of amebic dysentery, with its large, ragged ulcers and undermined edges, not infrequently resulting in perforation. The submucosa is

much thickened and infiltrated, the peritoneal coat is sometimes inflamed, and occasionally there are adhesions and hemorrhagic spots. Perforation seldom or never occurs. In the superficial layers of the inflamed region all sorts of bacilli and cocci are found; but in the deeper layers and in the adjacent inflamed and swollen mesocolic glands the *Bacillus dysenteriæ* is found in pure cultures.

When the small intestine is involved the changes are the same as in the colon, except there is not so much thickening of the submucosa.

The liver and spleen are not involved, while in the amebic type liver abscess is common.

MODE OF CONVEYANCE.

The disease does not appear to be directly contagious. The bacillus must be taken into the mouth, and this is accomplished ordinarily through the medium of infected drinking water. It may occur through infected milk or uncooked foods, and it is possible that it occurs from contamination by the dust from infected soil, and may be carried by flies in the same manner as in typhoid fever.

Intestinal disorders, indigestion, improper food, exposure to cold and wet, starvation, overcrowding, and, indeed, all bad hygienic conditions are strong predisposing causes.

CLINICAL HISTORY.

General Description.—The incubation period is very short, usually not more than two or three days, while in one case where a pure culture was intentionally swallowed, dysentery resulted within twenty-four hours. In another instance where a small quantity of a liquid culture was accidentally drawn into the mouth, the same result followed in less than forty-eight hours. In still another case where infection was supposed to have occurred through the rectum from the use of an infected chamber, the incubation period was twenty-four hours.

Ordinarily, the disease comes on suddenly. Often

the patient goes to bed well and wakes up in the night with pain in the abdomen, followed by diarrhea, the movements becoming more and more frequent, until they consist almost or entirely of blood and mucus. The number of movements varies from a dozen to fifty or more, and may be over a hundred in the twenty-four hours. There is more or less fever, intense thirst, much restlessness, coated tongue, straining at stool, and finally the movements become almost constant and involuntary. The swollen and infiltrated intestine can often be felt through the abdominal wall, and is very tender on pressure.

The disease is ordinarily of short duration, not more than ten days or two weeks. Before the end of the second week many cases will have terminated in death or convalescence, or the disease may gradually assume a chronic type.

Special Features.—The mode of onset is almost always sudden. The facial aspect in the early stages is anxious and expressive of suffering; toward the end it becomes apathetic. Fever is generally present, of irregular type and variable in amount, often reaching 103 F. to 104 F.; toward the end there may be a sudden fall to subnormal, a very unfavorable sign, or there may be a sudden rise. Rigors are rare. The skin at first is hot and dry, but toward the end it may be cold and clammy.

The pulse is rapid and often small and weak; death usually results from cardiac exhaustion.

The tongue is coated white, and at first is moist, but later becomes dry and glazed. Vomiting is unusual, though it does occur. Thirst is usually marked and persistent. The stools, at first diarrheal, soon consist of mucus and blood only; in the chronic stages there is an admixture of pus. Hemorrhage is rare, but does occur.

Abdominal pain is commonly marked, and there is tenderness over the seat of the lesions in the colon. The spleen and liver are not ordinarily enlarged or tender.

The blood changes are not very important. The white

corpuscles, especially the polymorphonuclears, are usually moderately increased, though they may be diminished, especially in the fatal cases. The red corpuscles, at first increased through the loss of fluids by the bowels, are later diminished on account of the loss of blood in the stools.

Pulmonary complications may occur, especially broncho-pneumonia. The nervous system is not markedly affected unless the lesions are high up in the colon or in the small intestine; delirium may occur toward the end of fatal cases.

The urine is often diminished in amount and retention occurs. Sometimes there is a small amount of albumen, and occasionally there is an acute nephritis. Parotitis is an infrequent complication.

Shiga states that the general symptoms are due to the absorption of the products of the solution of the bacilli themselves, and not to toxins produced by them; that these symptoms are not so marked as in typhoid fever is due to the location of the lesions in the less readily absorbing large intestine. Sometimes the lesions are higher up in the small intestine, and then the disease is much more grave and the toxemia marked. Symptoms such as high fever, headache, pains all over the body, sleeplessness, stupor, delirium, ecchymoses, etc., appear, and when the rectum is much involved, tenesmus is marked. The disease has a constant tendency to ascend, and the higher up the lesions the more marked the toxemia, the larger the stools and the greater the gravity of the case.

ASSOCIATION WITH OTHER DISEASES.

The older writers, particularly Manson, seemed to think that the disease is often associated with malaria; but this view is opposed by Strong as a result of his experience in the Philippines.

There have been, however, a number of cases of double infection with the ameba and with the Shiga bacillus.

There are two types, or rather stages, of the disease;

the acute form or ordinary epidemic dysentery of the tropics and temperate zones, and the subacute or chronic stages following the acute.

DIAGNOSIS.

Local rectal troubles causing blood and mucus discharges, such as hemorrhoids, polypus, malignant disease, etc., are to be excluded by direct examination. The most important differentiation is that from amebic dysentery, and this is often not possible from a consideration of the general symptoms alone. The most important points are the sudden and severe onset, the rapid course, high mortality, and epidemic occurrence of the Shiga dysentery, as contrasted with the more gradual and milder onset and course of the amebic disease and the endemic prevalence of the latter. The amebic type is, however, sometimes acute in its onset. Liver abscess seldom or never occurs in the bacillary disease, while it is common in the amebic form. In amebic dysentery, except when complicated with liver abscess, toxic symptoms, such as fever, malaise, headache, anorexia and nervous symptoms, seldom occur.

The specific tests are the only reliable ones; the isolation of the bacilli from the stools and the agglutination tests for the Shiga organism, and the finding of the ameba in the stools in the amebic type. The bacilli are found in the stools in all severe cases of Shiga's dysentery as early as the end of the first week in the acute stages, and sometimes in pure cultures. They may not be found in the later stages, and they are never present in the intestinal tract of healthy persons.

PROGNOSIS.

This depends on the severity of the epidemic and extent of the lesions. If the rectum and sigmoid flexure only are involved, the prognosis is favorable, while if the whole large intestine, and especially a portion of the small intestine is involved, it is very unfavorable. Nervous and other toxemic symptoms indicate such involvement, and are hence unfavorable signs. Failure

of the agglutination reaction to increase in intensity is also a bad omen.

The disease is always a grave one, the mortality ranging in the Japanese epidemics from 30 to 40 per cent. Recovery is rare in the more severe cases, and death usually occurs in the first ten days. Those who do recover are left very much weakened and emaciated, but usually without the digestive disturbances which so often follow amebic dysentery.

PROPHYLAXIS.

In the treatment the same precautions to prevent extension of the disease are required, as in typhoid fever; the most important of these consists in thorough disinfection of the discharges from the bowels and everything liable to have been contaminated by them, including the hands of attendants and instruments and appliances used about the patient. Personal prophylaxis includes also the use of water that has been sterilized and the avoidance of uncooked fruit and vegetables. The disease is a preventable one, and with military commands it should be prevented whenever it is possible to control the water and food supply of the men.

TREATMENT.

In the acute form the first essential is absolute rest in bed, with sufficient covering to protect from cold and draughts. Diet is also of the utmost importance, and should be exclusively liquid, and of such a nature as to be completely digested in the stomach. Milk is the best food, and may be given plain, peptonized or diluted with carbonated water or lime water; in the form of koumyss it is also well borne. All drinks should be tepid or warm, and given in small quantities at a time, and at frequent intervals. When milk is seriously objected to or can not be borne, strong broths of beef, mutton or chicken may be used; albumin water is of value in very severe cases. Stimulants may be given if needed, and the best is good brandy in the form of milk punch, or an occasional egg-nog.

The success which has been attained in Japan with the specific serum treatment instituted by Shiga gives that method the first rank in the consideration of therapeutic agents. Having had no experience with the method myself, I quote from Shiga's own article in the Twentieth Century Practice:

IMMUNIZATION OF HORSES AGAINST DYSENTERY.

An agar culture of the *B. dysenteriæ* which has been maintained at the incubator temperature for twenty-four hours is emulsified in normal saline solution and heated at 60 C. for one-half an hour. This mixture is then inoculated subcutaneously into horses in the usual manner, beginning with small doses which are gradually increased in amount. The immunization can be more easily produced if immune serum is added to the emulsion of the dysenteric bacilli, because abscesses which reduce the vitality of the animal are often produced at the site of the inoculation when bacilli alone are employed.

PRACTICAL SERUM TREATMENT.

Dysenteric serum, like typhoid serum, is bactericidal, but in practice the former gives much better results than the latter. Accrding to my opinion, this is due to the localization of the lesions and the toxic symptoms. In dysentery, the lesions are usually localized in the rectum and lower portion of the large intestine, whose physiologic function is principally mechanical, and where absorption of the food does not take place as in the small intestine. The toxic symptoms are intimately connected with this localization, against which bactericidal serum is valueless. Such symptoms in dysentery are happily less prominent than in typhoid fever. This is the chief reason for the favorable influence of dysenteric serum. This conception also accounts for the very unprofitable prognosis in enterodysentery.

RULES FOR ADMINISTRATION.

In accordance with my researches I have formulated the following rules for the administration of dysenteric serum for use in the Institute for Infectious Diseases in Tokyo:

1. In mild cases the serum is inoculated once in a dose of 6 to 10 c.c.

2. In cases of medium severity the serum is twice inoculated in doses of 10 c.c. The interval is from six to ten hours.

3. In severe cases the greatest quantities of serum are inoculated (about 60 c.c.). The daily dose does not, however, exceed 20 c.c.

By serum treatment the disease in its first stages is quickly cured or remarkably ameliorated. In advanced or inflamma-

tory stages the effects produced are about as follows: The number of stools is markedly diminished (generally reduced by halves for three successive days, followed by rapid recovery), the tormina and tenesmus decrease, the blood in the stools diminishes or entirely vanishes, the abdominal pains lessen, the fever vanishes, the appetite increases, and the general condition is much improved. In the ulcerative stages the action of the serum is not so pronounced as in the earlier stages, nevertheless healing of the ulcers with cicatrization often takes place. In such cases the fever is reduced, the number of stools is markedly diminished, the general condition is improved, and the appetite increases. In this stage even the results obtained are far better than those given by any other method of treatment.

By the serum treatment the course of the disease is shortened in those who recover, and lengthened in those who die. Of the 510 cases which I have treated (298 with serum and 212 with drugs) the entire course (counting from the beginning of the sickness to the discharge from the hospital) was on the average as follows:

	Recovered.	Died.
Serum treatment	25 days.	16 days.
Symptomatic treatment	40 days.	11 days.

The mortality of dysentery varies according to the climate, the epidemic, etc.; in order to estimate properly the value of the serum treatment I will here record the mortality in hospital treatment.

The mortality by the use of the serum treatment was reduced one-third below that given by symptomatic treatment, and was less than one-half the minimum obtained by such treatment.

MORTALITY IN HOSPITAL TREATMENT.

Year.	Place.	Treatment.	Number of Cases.	Deaths.	Percentage of Deaths.
1895	City Hospital	Symptomatic.	38	22	55.0
1896	City Hospital I.	"	221	86	39.0
1896	City Hospital II.	"	6	2	33.3
1897	City Hospital I.	"	336	143	42.4
1897	City Hospital II.	"	4	1	25 0
1897	City Hospital III.	"	767	304	39 6
1897	Inst. for Infec. Dis.	"	34	8	23 5
1898	City Hospital I.	"	178	73	42.0
1898	City Hospital II.	"	385	141	36.6
1898	Inst. for Infec. Dis.	Serum	65	6	9.2
1899	City Hospital I.	Symptomatic.	98	32	32.6
1899	City Hospital II.	Serum	88	11	12.5
1899	City Hospital III	Symptomatic.	481	105	22.0
1899	Inst. for Infec. Dis.	Serum	105	5	8.5
1900	Inst. for Infec. Dis.	"	40	5	12.5

Certainly this is a showing which encourages us to give the serum treatment a careful and extended trial. But Shiga says:

Whether or not serum-therapy is employed, suitable cathartics and enemata should always be administered, since these tend to limit the upward extension of the disease. For this purpose I generally order 0.5 grams of calomel, to be given twice, or 10 to 15 c.c. of castor oil, which is to be given but once. Thereafter, enemata of 1 per cent. saline or soda solution should be given once or twice daily. Combined with the serum treatment I generally order acid or alkaline drinks or red wine.

MEDICAL TREATMENT.

This may be considered under two headings, antiseptic and eliminative treatment. Of antiseptics, those usually employed are calomel, bichlorid of mercury and salol. All these have been highly praised and extensively used, but with the exception of calomel, which at times is very useful as an eliminative agent, it is doubtful if any of them have real value.

The most important eliminative agents used are the saline laxatives, especially the sulphates of soda and magnesia; ipecac in large doses is also highly recommended.

These remedies act first by emptying the bowels of all irritating fecal matter and then mechanically flushing them out, sweeping out at the same time many of the bacilli and their toxins.

Some physicians use the salines in large doses and only at the beginning of the disease, while others use small doses frequently repeated until blood and mucus disappear from the stools.

The ipecac treatment is very popular with the British, more especially in the British Indian service, but it has never gained much of a foothold among the profession either here or in our tropical possessions. In this method no food is given for several hours, then a mustard plaster is applied to the epigastrium and twenty or thirty drops of laudanum given by the mouth; half an hour after the laudanum two grams of ipecac are given in bolus or capsule, and the patient kept perfectly quiet with head low. If the medicine is rejected by vomiting it is repeated after an hour or two; if it is retained

it is nevertheless repeated after eight, ten or twelve hours, according to the effect produced, the object being to secure a feculent movement free from blood and mucus.

My own plan, as a result of experience gained in the tropics and the southern states, is to give 10 grams of Epsom salts in half a glassful of water every hour until a movement is obtained free from blood and mucus. This method is combined with irrigation of the lower bowel with warm normal saline two or three times a day, and as soon as the movements assume an ordinary diarrheal character astringents, such as bismuth subgallate in large doses, combined perhaps with a little Dover's powder, are given every three or four hours; should blood and mucus reappear, the saline flushing is repeated. The lower down in the bowel the lesions are seated the more important becomes the local treatment by irrigation.

In convalescence great care is required in the management of the diet. These patients are usually greatly weakened and emaciated, and a slight indiscretion in regard to food will often cause a serious relapse. Only liquids or very soft and digestible solids should be used for some time after the movements become normal. A change of climate or a sea voyage greatly hastens recovery, and for patients in the tropics is often essential.

TREATMENT OF THE SUBACUTE AND CHRONIC STAGES.

In the subacute and chronic stages of the disease the treatment is somewhat different, but even here salines may often be used with advantage in the beginning. Local treatment is, however, of the first importance in these cases; the patient should be given an anesthetic, the sphincter well stretched, and the rectum carefully examined with the speculum to detect ulcers. These ulcers will often be found with a little pocket at the lower end in which fecal or other foreign matter lodges, keeping up the irritation. These pouches must be slit up to the bottom, scraped with the curette, or touched

with pure carbolic acid or lunar caustic; after this the use of warm saline injections two or three times a day with proper diet suffices to effect a cure. When the lesions are higher up nitrate of silver injections, 1 to 500 or 250, are given every day or every other day; the injections should be large, two or three pints, warm, and given slowly through a tube introduced 12 or 15 inches; the patient should be in the left lateral position with the hips raised. Internally, large doses of dermatol are useful in these cases.

A CONTRIBUTION TO THE STUDY OF THE SUMMER DIARRHEAS OF INFANCY.

J. H. MASON KNOX, Jr., Ph.D., M.D.,
Assistant in Pediatrics, The Johns Hopkins Medical School; Physician in Charge of the Thomas Wilson Sanitarium, Mount Wilson, Md.
BALTIMORE.

It is with great hesitancy that any attempt is made in this paper to enter a field that has been so thoroughly investigated during recent years as that embraced in the title of "The Summer Diarrheas of Infancy." The zeal with which these intestinal disorders of children have been studied lately is in direct ratio with their neglect by early medical writers. Although, as Dr. Booker[1] has shown, there are occasional references among the ancients to diarrheal troubles in infants in the summer months, there is no clear history of these diseases prior to their appearance among the colonists in this country. As he suggests, the importance of human life in this newly-settled land may have called sharp attention to a malady which was each summer carrying away the children of the settlers in large numbers. The malady, called the "dread of parents and the opprobrium of physicians," was thought at first peculiar to America. The yearly outbreaks in the larger towns scattered along the Atlantic coast were recognized and described under various names by local physicians, but the first adequate description of these maladies seems to have been made in 1773 by Dr. Benjamin Rush.

1. Arch. of Pediatrics, xviii, 1901, p. 487.

Knowledge of these disorders spread gradually in this country and in Europe, and it was not until the early part of the last century that their great importance was recognized. As is the case with other diseases, the clinical descriptions of many of these early observers leave but little to be added by their successors. They ascribed the causes of the disorders to various conditions, some of which will pass muster to-day, to the character of the atmosphere, cold, moist or hot, to direful miasms, to overcrowding, to improper food, to teething, to faulty action of the liver, and so on. They recognized the epidemic character of the disease and the lines of treatment adopted were in the main those now followed. Food was restricted, prompt purgation resorted to, opium was used in moderation, and the advantage of removing the little patients from crowded districts to the country was recognized. With the advent of the science of bacteriology, new zest has been added to the study of these complaints. Then, for the first time, there seemed possible a rational explanation of the infectious nature of the affection. The intestinal tract of the healthy infant was found to swarm with bacteria of many varieties.

It would be needless to even mention the host of workers in this field, or the names of the numerous bacterial forms isolated. Perhaps the most authoritative work on the subject has been done by Escherich of Vienna, formerly of Gratz, who noted the predominance of *B. lactis aerogenes* in the upper bowel of the infant, and of *B. coli communis* in the large intestine, during health. In diseased conditions the differences between the flora in the upper and lower bowels are less marked. In addition to other varieties found in the intestinal tract, including the interesting *B. acidofilus* of Moro and Finkelstein, several forms known to be pathogenic for man have been isolated from the stools of infants suffering from diarrhea. Thus, among others, the pathogenic cocci, *B. proteus vulgaris, B. pyocyaneus* and virulent forms of the colon bacillus have been described. No single patho-

genic organism, however, has been found constantly in these diarrheal conditions, and the conclusions of Dr. Booker[2] perhaps voice the present position of the profession, that "No single organism is found to be the specific exciter of the summer diarrheas of infants."

Notwithstanding these comparatively negative findings, it is believed that there is clinical evidence in favor of the specific nature of these affections. The epidemic character of the disease in our crowded cities during the summer months, its occasional propagation in hospitals and asylums from patient to patient, and the nature of the illness itself, all suggest that the exciting cause of the disorder is an infectious agent not normally present in the body. The factors predisposing to this infection have long been recognized, and experience only tends to emphasize their importance. Thus the disease is much more prevalent among infants of weakened constitutions living amid unhygienic surroundings and nourished by insufficient or improper food. The high temperature during our summer months provides most favorable conditions for the rapid proliferation of bacteria in food products, especially in milk, and the large residue of undigested milk which remains in the infant's intestinal tract as a result of immoderate feeding, or of a weakened absorptive power, forms an ideal pabulum for the growth of bacteria and the formation of deleterious products. So frequently are these conditions incident to an attack of summer diarrhea that many, including Czerny of Breslau, consider improper feeding the more important factor in its production, and the infective element to be of comparatively less moment. Certain it is that the mortality in intestinal diseases of infants is directly influenced by the resistance of the patient, which is in turn dependent upon the condition of the digestive apparatus.

The effort to isolate a single organism which could be shown to be the cause of a large group of these cases of

2. Johns Hopkins Hospital Reports, vi, p. 251.

summer diarrhea, has been unsuccessful, because of the difficulty in the separation of such an organism, if present, from the great numbers of bacteria in the intestinal contents. Last summer in the laboratory of the Thomas Wilson Sanitarium,[3] assisted by a grant from the Rockefeller Institute, two investigators, C. W. Duval and V. H. Bassett, succeeded in isolating *B. dysenteriæ* (Shiga) from the stools of forty-two infants suffering with diarrhea. The importance of this finding is evident, as it brings a series of diarrheal cases in young children into relationship with an organism that is the proven exciting cause of certain varieties of severe intestinal disease in adults. It is unnecessary to do more than refer to the history of the organism. It will be remembered that it was first isolated by Shiga[4] in Japan, in 1897, from the stools of patients suffering from epidemic dysentery. In 1900 Flexner[5] discovered the same organism to be the cause of many cases of epidemic dysentery among our soldiers in the Philippines. Soon after Kruse,[6] in Germany, found the same bacillus in the dejecta of patients suffering from so-called "asylum dysentery." It has since been successfully isolated by Vedder and Duval[7] in epidemics of dysentery in this country, and has also been found in diarrheal cases in China, in Constantinople, and in several other sections of Europe.[8]

At the Thomas Wilson Sanitarium the stools of about fifty-three patients, most of them under 1 year of age, were examined bacteriologically, and from forty-two the dysentery bacillus was isolated. Early in the summer those cases were selected in which the stools contained

3. The Thomas Wilson Sanitarium for Children is situated in the country, ten miles from Baltimore, and receives during the summer months upwards of 400 children suffering from summer complaint.

4. Centralblatt für Bakteriologie, 1898, No. 24.

5. Philadelphia Med. Jour., vi, 1900, p. 414.

6. Deutsch. Med. Wochenschrift, 1900, p. 637.

7. Journal of Experimental Medicine, vi, 1902, p. 181.

8. Pfahl. Zeitschrift f. Hygiene u. Infectionkrankheit, 1902, Bd. 40.

considerable mucus and blood. These were regarded as especially "favorable," that is, that from them the organism could be obtained in largest numbers. Later the stools of twenty-five consecutive infants were carefully plated, and in nineteen instances *B. dysenteriæ* was found. All these babies exhibited some form of intestinal disorder, but the stools of many of the series were largely fecal, and in these cases the organism was present in comparatively small numbers, and was more difficult to isolate. The importance of determining the presence or absence of the bacillus in normal stools was recognized, and the discharges of twenty-five babies, ranging in age from 7 weeks to 2 years, suffering from no intestinal derangement, were subjected to painstaking examination. In none of these cases was the Shiga bacillus found.

A detailed account of the methods employed in isolating the organism will shortly appear,[9] and will be only briefly referred to here. The technic was essentially that used by Dr. Flexner in the Philippines, though several important modifications employed first by Vedder and Duval[7] in their work on adult dysentery, were of great assistance. The stool of the infant to be investigated was collected on sterile gauze and taken immediately after passage to the laboratory. Numerous plate cultures in acid agar-agar were made, choosing for plating, when possible, bloody or muco-purulent portions. After incubation for twenty-four hours, the superficial colonies were marked off and the plates again placed in the thermostat for twenty-four hours, when the new superficial colonies were carefully examined. Duval and Vedder found in their studies that the dysentery bacilli grow out on plates less rapidly than do colon organisms, and that by thus marking the plates one is enabled to select for study the colonies developing more slowly, and thus avoid the ubiquitous colon bacillus. The colonies which seemed morphologically favorable after this second incubation were transplanted to glucose agar and all

9. Journal of Experimental Medicine.

gas-forming organisms discarded. The non-gas-forming bacilli were studied culturally on the various media, and their agglutination reaction tested with serum from the same patient, the sera from numerous other patients, and with antidysenteric serum furnished through the kindness of Dr. Gay. This agglutinating action of the blood serum for the specific organism was a great assistance in the successful isolation, as it proved to be to Shiga and Flexner, and to all subsequent investigators. The technic of making this test with the dysentery bacillus requires more than usual skill, and thus far wider variations in the amount of agglutinin in the sera from various patients seem to pertain than is the case in typhoid fever. Our experience would indicate that in nearly all cases the serum has some definite agglutinating power for the specific organism if the two are brought together under the most favorable conditions. The serum from immunized horses in dilutions from 1-100 to 1-1,000, agglutinated the organisms isolated from all our forty-two cases, as well as various strains of the dysentery bacillus sent from abroad, while it was quite inert in low dilutions against the typhoid bacillus. Moreover, the blood of our patients agglutinated the organisms isolated at the sanitarium, as well as those obtained from cases of epidemic dysentery in Japan, the Philippines, Germany and in this country, in dilutions varying from 1-16, the least active, to 1-1,000, when cultures of typhoid or colon bacilli tested synchronously were negative. The agglutination reactions were made in drop slides and observed under the microscope. Lenz,[10] in the last few months, has advocated the macroscopic test-tube reaction as being more reliable, and since last summer this method has been used in testing many of our isolations. With some minor differences, the two methods seem to fairly correspond. It is difficult at present to explain many apparent irregularities in the agglutination reaction with the dysentery bacillus, and

10. Martini u. Lenz. Zeitschrift f. Hygiene, xii, p. 2, 1902.

much further study is necessary before hard and fast lines can be drawn as to the time in which a reaction must take place to be considered positive, and also as to the minimum dilution of the serum to be allowed. These steps toward a recognized technic were gradually taken in the study of the Widal reaction with the typhoid bacillus. However, the Shiga bacillus seems to present more differences in the rapidity with which the various isolations clump than does the typhoid bacillus. In no instance during the summer did normal blood agglutinate any of the dysentery bacilli.

It is too early yet to speak positively of the clinical value of the agglutination reaction. Of thirteen cases tested during the first week of illness, ten gave positive, and three negative, reactions. One case reacted on the second day. Usually, however, reaction was more marked later in the disease. In the chronic cases the blood may remain positive for weeks or months. It is in cases of this nature that the blood tests may prove useful. By this means in a number of our series presenting the symptoms of chronic intestinal disorder, a diagnosis was made on the basis of a positive blood reaction, and a dysentery bacillus afterward isolated from the stools. In several tested cases where the bacteriologic examination of the stools was not possible or convenient, a diagnosis was ventured because of an undoubted agglutinating reaction. It is hoped that when its limitations are understood this blood reaction will prove of assistance to the clinician.

The studies conducted by Duval and Bassett have resulted in establishing the fact that there was present in the intestinal tract of a large number of infants suffering from diarrheal disorders, a bacillus identical with the *B. dysenteriæ* (Shiga), hitherto associated only with the dysentery of adults. They have found also that this organism was pathogenic for animals; that it was present in largest numbers in the deeper layers of the intestine, as shown by scraping the mucosa at autopsy; and that

the number of bacilli in the stools was in proportion to the acuteness and severity of the symptoms. The organisms isolated were agglutinated by antidysenteric serum and by the blood of infected patients, never by the blood of those ill with other diseases. They were unable to find the Shiga bacillus in the discharges of healthy infants. In short, the evidence presented by Shiga, Flexner and others, to establish the etiology of dysentery in adults is at hand to show that the dysentery bacillus is also the causal factor in certain diarrheal diseases among children.

It now remains to consider the clinical aspect of this series of cases in order to determine, if possible, whether they belong to an isolated group, or whether they represent in symptomatology and pathology the maladies called by the descriptive epithet, "The Summer Diarrheas of Infants."

TIME OF ADMISSION.

The 42 cases of our series were admitted to the sanitarium between June 13 and August 21 of last summer, 12 being received in June, 25 in July and 5 in August.

AGE.

The age of the little patients varied between 7 weeks and 4 years. The majority of them were under 1 year of age. They may be arranged according to age in the following periods of six months each:

Under 6 months	13
Between 6 months and 12 months	17
Between 12 months and 18 months	8
Between 18 months and 24 months	3
Over 2 years	1

The interest of this classification is that our patients, in regard to their ages, correspond closely to a period in infancy long known to be most susceptible to summer diarrhea.

SEX AND RACE.

The cases were about equally divided between the sexes. But two colored infants are included in our series, as the accommodations for these patients are limited.

HISTORY OF ILLNESS.

Unfortunately, because of the distance of the sanitarium from the city, the patients usually came under our observation after their initial symptoms. The mothers, as a rule, however, gave us a fairly definite history of onset, the record often being that the baby, after a day or two of languor, was suddenly seized with diarrhea, usually preceded or followed by vomiting. At the same time a rise in temperature was noted. Subsequently there was increased constitutional disturbance indicated by a loss of appetite, malaise and restlessness. The stools at onset, although increased in number and often fluid in consistency, were fecal in character. Soon, however, except in the very mild attacks, the fecal odor and appearance were lost, and the discharges were composed largely of greenish mucus, and not infrequently tinged with blood. During the height of the illness the temperature usually continued elevated, and nausea and vomiting, even after a reduced diet, were common. It was at this period that the Shiga bacilli were obtained in large numbers in the stools. These acute attacks varied greatly in severity. In some cases the constitutional disturbance was slight and the stools were little affected; on the other hand, several of our patients presented early symptoms of profound intoxication, evinced by convulsive spasms, excessive nausea and vomiting, and increased languor, ending in coma. A large number of the cases, however, exhibited quite a different picture. After an acute onset, instead of toxemia ending fatally or in a prompt convalescence, the baby showed symptoms indicating a more localized affection of the intestine. In these instances, which included more than half of our series, after variable periods the stools became mucopurulent, and contained often an increased amount of blood, some of it bright red in color. The temperature showed a daily, usually an evening, rise. The appetite remained poor, and vomiting was not infrequent. There was evidence of considerable pain in the lower abdomen;

tenesmus was marked. These cases often became chronic and lasted for weeks. They were more numerous during the latter part of the summer, and were less amenable to treatment. Often, despite many carefully prepared diets, there was progressive loss of weight, increase in anemia, and a fatal termination from exhaustion. In several instances in our series the specific infection was superimposed on infants which for months had been suffering from malnutrition and chronic intestinal indigestion. Of several of the clinical features a few words may be added.

TEMPERATURE.

The temperature in the series of cases varied from 95 F. to 105 F. As has been mentioned, daily rises in temperature were often noted. The degree of bodily heat was of clinical assistance only when considered with other signs and symptoms, as a drop to normal or subnormal temperature not infrequently was an indication of collapse, and the illness of several of our patients manifesting constitutional disturbances, ran an almost afebrile course.

GASTROINTESTINAL PAIN.

Gastrocolic was rarely noticed while at the sanitarium, but was present, according to the description of the mother, in a few cases at onset. A large number of the cases of long duration exhibited rectal tenesmus.

NUMBER AND CHARACTER OF THE STOOLS.

The stools varied in number from one to thirty in twenty-four hours. They usually became most frequent in the first three days after onset. The discharges presented also wide differences in character. Some were distinctly fecal, but semi-fluid or fluid in consistency. In the cases showing symptoms of indigestion, the stools were often pasty in character and gray in color. Hard masses would alternate with softer material. The element that was almost constantly present was mucus, either as a firm, translucent, gelatinous substance, or as

more fluid, viscid, or stringy material, usually greenish in color. Marked inflammatory lesions of the lower bowel were indicated by the muco-purulent or purulent character of the stool. The amount of blood passed varied from a slight tinge or fleck to dark brown or bright red masses in fairly large quantities. Under the microscope many epithelial cells, a varying number of white and red corpuscles, bacteria, mucous strands and undigested food residue, fat globules and casein, composed the more constant elements.

COMPLICATIONS.

Complications early in the course of the illness were comparatively infrequent. Death in several instances was preceded by the appearance of livid erythematous blotches over various parts of the body. Terminal bronchopneumonia was not uncommon. In two instances there was concurrent lobar pneumonia, and in two other cases acute parenchymatous nephritis developed. Occasional edema of the extremities was noticed not associated with alterations of the kidneys, and in several instances stiffness of the neck and extremities would have suggested meningitis had it not passed away after a few hours.

ILLUSTRATIVE CASES.

A résumé of each of the histories of our series would hardly be profitable, as in the main they present very similar and familiar features. From a large number, however, several may be selected as representing the more important clinical groups. The following is an instance of a mild form of diarrhea:

CASE 1.—A child 18 months old, fed at the table, became cross and irritable. A few days afterward its stools increased to from seven to ten a day and were described by the mother as "green and slimy." On admission to the sanitarium they were found to consist largely of green mucus, with numerous soft, white lumps. They never contained any blood. There was occasional vomiting. Physical examination, negative. There was no marked prostration. On a regulated diet the discharges became homogeneous in form, the mucus disappeared, and the child soon completely recovered.

A much severer type of an acute case with complication is illustrated by the following instance:

CASE 2.—An infant 8 months old, fed on condensed milk and previously well, was suddenly seized with moderate diarrhea. For several days the stools were not more than six in twenty-four hours, but on the day before admission they increased to twenty, and were fluid, with considerable mucus, but no blood. The general condition of the baby became markedly worse. He was feverish, restless and had several fainting spells. On admission the child was in collapse. The rectal temperature was 105 F., pulse 180, face pale, and extremities cold. The eyes were sunken and the abdomen scaphoid. Physical examination of the chest, negative. There was some response to stimulation, and for two days his condition improved. On the third day, however, albumin in large quantities appeared in the urine, with many granular casts and pus cells. The tissues about the neck and chest became edematous; the child gradually became more listless; coma ensued, ending in death three days after the renal involvement. At autopsy the intestines were little altered. There was no distension. The mucosa was pale. Peyer's patches were moderately enlarged and somewhat injected, as were also the solitary follicles. The mesenteric glands, particularly those draining the sigmoid flexure and rectum, were somewhat enlarged. There was no lesion of the intestinal mucosa. The kidneys were the large and white kidneys, indicating acute parenchymatous nephritis.

In the next case, although not unlike clinically, there was evidence of a destructive lesion of the lower bowel.

CASE 3.—An infant of 5 months, fed for four months on condensed milk, began two weeks before admission to be fretful and to vomit occasionally. Soon after the stools increased in frequency and were described as "yellowish and slimy." No blood was noticed. The child was found, on examination, to be rickety and anemic, and did not improve after admission. There was a moderate fever. The stools were six to seven a day, and consisted of thick, viscid mucus and contained considerable blood and pus. There was some rectal tenesmus. The baby gradually lost weight and strength, the blood in the stools increased, and death ensued about five weeks after the onset of acute symptoms. At autopsy numerous small hemorrhages were found scattered through the ileum; the mucosa of the entire colon was thickened and covered with bloody mucus; the solitary follicles were much enlarged and the seat of numerous small ulcerations. The mesenteric glands were everywhere swollen and red.

One other history will be cited as illustrative of a case of long-standing malnutrition.

CASE 4.—A patient was 7½ months of age, a twin and delicate from birth, breast-fed for two months, but during the remaining five months the diet has been variously modified. For most of this time the stools varied from two to eight in twenty-four hours and frequently contained mucus. There was little or no gain in weight. Frequent colic; occasional vomiting. The abdomen was usually distended. Two weeks before admission these symptoms became somewhat aggravated. The child was found on examination to be markedly rickety, with general glandular enlargement. After admission its general condition remained about the same. For days at a time the stools would become more homogeneous and firm, only to show a return of mucus and pus after a short period, despite careful feeding and rectal irrigations. There was never any blood. The temperature was but slightly elevated. The infant remained with us until the end of the season, and was discharged unimproved.

What has been said of the clinical history of our forty-two patients and the illustrative cases cited, are sufficient, certainly, to warrant the assertion that, so far as can be told at the bedside and in the autopsy room, we were dealing last summer with no unusual form of intestinal disease, but with the common varieties of summer diarrhea. Our cases fall naturally into two or three large groups which have long been recognized, namely: into those of an acute dyspeptic or fomental diarrhea (gastrointestinal catarrh) of varying degrees of severity, in which the toxic symptoms were prominent—twelve cases; into those of colitis, marked by the definite inflammatory lesions of the intestinal mucosa—twenty-six cases; and again into at least four instances which were primarily malnutrition, marasmus, or chronic indigestion, but in addition to which the element of infection has been added. It is well understood that these divisions of intestinal diseases do not represent definite clinical entities, but that one form of illness may readily pass into another; that the cases of ileocolitis usually begin with symptoms of dyspeptic diarrhea, and that ill-nourished infants are particularly prone to either form of disorder. Of the most fulminating variety of the toxic intestinal diseases, namely, cholera infantum, we had no example in our series. It is suggestive, however,

that in the stool of an especially acute case, one thought at first to be cholera infantum by the attending physician, the Shiga bacilli were found in greatest numbers. It will be reiteration to go over the histories of the infants treated in previous years at the sanitarium. In the summer of 1901 nearly five hundred intestinal cases were admitted. These, too, can be roughly divided into the groups just indicated. During that season we were able to obtain more than twenty autopsies. The findings at the postmortem table substantiated those of the cases of our present series which came to autopsy. The intestines of infants observed two years ago suffering from acute toxic affection likewise presented little pathologic alteration. The mucosa was pale and thin, the lymphatic elements in the intestinal wall moderately enlarged, and the mesenteric glands swollen. On the other hand, in a second group of cases the lower bowel was extensively involved, the mucosa being thickened, and often the seat of more or less extensive ulceration, the ulcers beginning usually at the summit of the swollen solitary follicles. The mesenteric glands were hypertrophied. It is an interesting fact that neither in our autopsy findings of last summer nor in the postmortem examinations of the larger series of 1901, was any false membrane found lining the intestines, as is so frequently described in the acute epidemic dysentery of adults.

LEUCOCYTE COUNT.

During that summer the leucocytes in a large number of intestinal cases were examined by Dr. Warfield and the writer.[11] A leucocytosis was found, but the number of white cells varied within such wide limits, even in the mild forms, that a high or low count could not be regarded as of diagnostic value. The differential count, however, showed that while there was no change in the relative ratio of the white cells in the normal infant, in the simple diarrheas, or in malnutrition, that as the case be-

11. Johns Hopkins Bulletin, xiii, p. 186.

came more severe in type there was a progressive increase in the polymorphonuclear neutrophilic cells and a decrease in the small mononuclear variety, and that this polymorphonuclear leucocytosis took place both in the cases of acute intestinal poisoning and in the more severe forms of ileocolitis. The counts made last summer in the series of cases under consideration confirm these results and add some support to the contention that similar varieties of intestinal derangement were under observation.

TREATMENT.

The treatment pursued at the sanitarium followed the usual lines, and will be but briefly referred to. In the acute cases, milk was withheld and a cereal water with thin broths, or plain water with a little egg albumin, substituted. Whey or a whey-cream mixture was found a useful intermediary step to the gradual return to a milk diet. With the chronic cases, milk, beginning with high dilutions, occasionally predigested, often with a cereal water as a diluent, formed the chief reliance. Colon irrigations were frequently given and gastric lavage as the symptoms indicated. Drugs were sparingly used. An initial purge of calomel or castor oil was administered when the baby came early under observation. Various astringent preparations, tonics and opiates were occasionally employed. Injections of salt solutions under the skin were less frequently resorted to than in former years, as it is felt that this procedure has been somewhat over extolled. As stimulants, strychnin, digitalin, alcohol, and atropin were found serviceable in the order named. In the giving of stimulants we were greatly helped by the introduction by Dr. H. W. Cook,[12] clinical assistant, of a modification of the Riva-Rocci sphygmomanometer, by which the blood pressure of the little patients was rapidly recorded. With the additional aid

12. The full description of this work appeared in Amer. Jour. Med. Sci., cxxv, 1903, p. 433. The mechanism is described in THE JOURNAL A. M. A., May 2, 1903, p. 1199.

of this instrument at the bedside, Dr. Cook shows that the actual vitality of the infant can be more accurately determined than by the skilled finger on the pulse, and the kind and amount of stimulant suited to the particular need of the hour. Late in the summer we were supplied by Dr. Flexner with an anti-dysenteric serum found protective to laboratory animals. Unfortunately, at this time there were no acute cases at the sanitarium. Several infants ill with ileocolitis, from whose stools the Shiga bacillus had been isolated, were injected hypodermically with 5-10 c.c. of this serum twice a day. No change due to the serum could be noted in their condition. No untoward effects followed the serum treatment, and it is hoped that more powerful serum in perhaps larger doses will prove to be of therapeutic value, particularly in the early cases.[13]

Enough has been said to make probable the assertion that the infant population of Baltimore during the summer of 1902 suffered from no unusual scourge, but rather was subjected to the accustomed epidemic of diarrheal diseases regularly recurrent during the warm weather, and that typical instances of these disorders were under treatment at the Thomas Wilson Sanitarium. That these infants were not the victims of an infection local to the sanitarium is shown by the fact that they were admitted from all sections of the city already ill. Two cases were from more distant parts of the state. Moreover, since the results of the investigation by Duval and Bassett have been known, other workers have found the dysentery bacilli in the stools of babies suffering from diarrhea in other localities; thus, Zahorsky,[14] in St. Louis, and Park and Dunham, in a small epidemic at Seal Harbor, Maine, have reported such cases. The extent of epidemics in Japan and in the Philippines indicates that the organism is capable of very wide distribu-

13. Moser, in Jahrbuch für Kinderheilkunde, vol. lvii, 1903, p. 8, reports gratifying results in scarlet fever cases from the injection of 180 c.c. of an immune serum.

14. St. Louis Courier of Medicine, Dec. 1, 1902.

tion. Only future research can determine the geographical or climatic limitations of the infection, but it would appear that a large group of the summer diarrheas of infancy are associated with bacilli identical with, or closely related to, *B. dysenteriæ* (Shiga). Recent work by Lentz,[10] His and Russell,[15] Park,[15] and others indicate that minor differences may exist in the cultural properties and startling variations are pointed out in the agglutination reaction of the bacilli isolated from different sources. Thus Park finds that in indol and acid productions, as well as in agglutination reactions, the Japan and New Haven cultures are separated from the Manila, Baltimore, New York and Seal Harbor cultures. Whether, in the search for the cause both of dysentery in adults and of summer diarrhea in infancy, one has to deal with a group of very closely related organisms, or, as is more probable, with slightly different strains of the same bacillus comparable to the differences that can be shown in the various typhoid and colon cultures, is a most interesting and important problem for the bacteriologist to determine.

CLINICAL SIGNIFICANCE OF THE FINDING.

This bacteriologic finding in connection with the summer diarrheas of infancy is of no less interest to the clinician, as it spurs him on to attack with renewed vigor the old problems of prophylactic treatment. Professor Koch,[16] in his notable address two years ago in London, showed the blunder of attempting to treat pestilences according to a general scheme. He said: "Every disease must be treated according to its own individuality, and the measures to be taken against it must be accurately adapted to its specific nature, to its etiology." If the investigations of last summer are substantiated, effort may be directed for the first time against a recognized foe. Questions, then, of the

15. New York Medical Record, vol. xiii, Feb. 28, 1903, p. 357.
16. American Medicine, vol. ii, Jan. 27, 1901, p. 145.

utmost importance can be propounded with some confidence that they will be accurately answered.

METHOD OF PROPAGATION.

One of the first of these problems is to determine the means by which the infectious agent reaches the infant. Although the number of cases in our series is too small to warrant positive assertions, there is some rather suggestive data on this point in the histories of our patients. It can hardly be doubted that the disorders being considered primarily affect the gastrointestinal tract, and therefore that the mouth is the portal of entry, and that the source of infection takes place in the great majority of cases through food or drink, that is, in young infants, through contaminated milk or water. Condition of the milk supplied to the poor from the small stores in our large cities is notoriously bad. During the summer of 1901 Bassett and the writer[17] examined a large number of samples of milk supplied the babies in Baltimore suffering from summer complaint before their admission to the sanitarium. This milk was found to have an average germ content of over four million to 1 c.c., and several samples contained ten to twenty million bacteria in each cubic centimeter. Last summer Bassett and Schorer[18] repeated and verified this work. They were able to show a progressive increase in bacteria and in diluents in the milk during each stage of its handling from the farmer through various dealers to the baby. Because the milk is so good a culture medium in the warm weather without proper refrigeration, a small initial contamination is sufficient to render it in a few hours unfit for infant feeding.

For the explanation of the sudden outbreaks of this illness at the same time in different sections of the community, one is tempted to turn to a general source of infection. The dysentery organism is related bio-

17. Maryland Medical Journal, June, 1902.
18. Maryland Medical Journal, July, 1903.

logically to the colon and typhoid bacillus which are usually conveyed to man through water.

With this thought in mind, an effort was made to find out the character of the water supplied to our patients. The mothers were asked specifically whether the water used in preparing the nourishments, and what is just as important, that supplied to the infants between feedings, was boiled. Concerning only twenty-eight out of the forty-two babies was fairly reliable information obtained as to both the milk and the water used. In twenty-six of these cases unboiled city water was given the babies, either with their nourishment or separately, and in but two instances was all the water supplied boiled. Of particular interest are four infants ill with this specific diarrhea, who were exclusively breast-fed, but to whom unboiled water was given between nursings. Four other babies whose only food was condensed milk, were likewise given unboiled water between feedings. These cases are too few to admit of generalization, but they do suggest that the dysentery, like the typhoid bacillus, may be harbored in the public waters. Indeed, it is possible that infection of the milk with the organism is brought about through dilution with impure water. It is possible to conceive that the disease may be propagated in other ways. Outbreaks of this form of summer diarrhea in asylums and hospitals, where sterilized milk and boiled water are used, are probably brought about by the spread of the disease from patient to patient, through carelessness on the part of attendants. Another source of danger is insects, particularly the common house fly. In the only case in our series contracted at the sanitarium infection was probably conveyed through flies. The child concerned was fed exclusively on pasteurized milk, and given only sterilized water, but there was no means at hand to adequately protect him from the swarms of flies, which may easily have carried the infective agent from a napkin to his hands or feeding bottle.

It is difficult to account for the comparative freedom of infants from this form of illness during cold weather and its regular return as a widespread epidemic in summer. Shiga has pointed out that the dysentery bacillus is kept alive in Japan by the occasional sporadic cases which occur during the winter in that country. Evidence suggesting a similar condition among infants in America is accumulating. Thus, in four children brought to the Johns Hopkins Hospital because of diarrhea during January, February and March of this year, the Shiga bacillus was isolated by Bassett, and Ford has lately found the same organism in the stools of a man suffering from a mild gastro-enteritis. By means of such sporadic cases, and because this bacillus may be harbored for months in small numbers in the intestines of chronic cases, it is not destroyed during the winter, but is at hand each summer to rapidly proliferate under favorable conditions. It has been repeatedly emphasized that infants suffering from marasmus, less severe forms of malnutrition, and from other chronic ailments, are its readiest victims.

The name, "dysenteria infantum," or "infantile dysentery," is suggested for the infection under consideration. This designation would distinguish these cases from other forms of intestinal disorder, and also from the specific dysentery of adults, which, although having the same etiology, seems to present clinical and pathologic differences.

From the foregoing discussion, it may be fairly concluded that:

1. The diarrheal disorders of a large series of children treated in the Thomas Wilson Sanitarium during the summer of 1902 were produced by a bacillus thought to be identical with the *B. dysenteriæ* (Shiga).

2. The cases in this series presented the clinical and pathologic features of the several forms of the summer diarrheas of infancy.

3. There is good reason for the confidence that a pro-

portion, and probably a large one, of the so-called summer diarrheas of infancy is caused by the *B. dysenteriæ* (Shiga).

4. A confirmation of the work of Duval and Bassett by establishing the etiology of this yearly epidemic among infants, will make possible the use of more intelligent measures to control and suppress it.

5. More success can be expected from prophylactic measures and from medicinal and serum therapy than ever before.

MALARIAL DYSENTERY.

WILLIAM BRITT BURNS, M.D.
MEMPHIS, TENN.

One has but to review the literature of dysentery to arrive at the conclusion that there is no fixed etiologic factor in this disease; that the lesions, which are mainly confined to the large intestine, are quite characteristic, but are induced by several different agents, is a pretty constant finding, and is worthy of consideration.

The *Amœba coli* is designated as the specific causative factor of dysentery, but here one is confronted with the fact that these bodies are found in less than one-half of the cases of all varieties and stages of the disease, and that the colon may be the normal habitat of amebæ, not only of this, but other amebæ—*Amœba guttula, diaphana, vermicularis, oblonga, reticularis and proteus*—which, it may be, are quite as culpable as *Amœba coli.* Moreover, these bodies are usually accompanied by other organisms, some pathogenic, and others which, by the proper environment, may be made pathogenic. Again, injections of amebic stools into the bowels of laboratory animals have not produced dysentery, and on the other hand, ameba-free stools injected in like manner have produced the flux. *Amœba coli* occurs nearly constantly in the pus of about half of the cases of liver abscess from dysentery; bearing out the contention of Councilman and Lafleur that amebæ are found, not alone in the pus and on the walls of intestinal ulcers, but in the tissues around them, they could not, therefore, reach the liver without some such portals of

entry. I quite agree that the *Amœba coli* is one of the agents in the etiology of dysentery.

The work of Shiga, Flexner and others have given the profession a ray of hope looking to the minimizing of the agents of this great scourge, but up to the present we are only able to say that there are "forms of dysentery," under which heading "several diseases unlike in their etiology and pathologic anatomy and, occurring under different conditions of climate and locality, although resembling each other to some extent in their symptoms." Thus we have dysenteric and diarrheal symptoms accompanying typhoid infection, pneumonia, bronchitis, hepatitis, abscess of the liver, etc.

Celli and Fiocca believe that the *Bacterium coli communis* is the etiologic factor in dysentery; this in connection with a bacillus resembling the typhoid bacillus and streptococci induce dysentery. Flexner has observed in the Philadelphia Hospital one case of dysentery, where the *Bacillus dysenteriæ* was found, and states that further observations are necessary to determine definitely whether this form of disease prevails in this country.

In short, it seems the part of wisdom to admit that in so far as there is a distinct, definite causative factor in the production of the fluxes, we are largely in doubt.

Thus, desultorily, I am lead to the subject of this paper. I could not better begin than by quoting from Manson: "In respect to geographic distribution and certain epidemiologic points, tropical dysentery bears a close resemblance to malaria. Like malaria, although dysentery is found in temperate climates, in them it is, with exceptions, a comparatively rare and mild affection. Like malaria, as we approach the Equator dysentery tends to become more common, and in type more severe. Like malaria, it is endemic in certain spots, where it often appears in sporadic form, and, at times, in limited local epidemics. Like malaria, it may extend beyond these endemic spots and involve more extensive tracts

of country. Like malaria, it appears to have a predilection for low-lying damp places; and, like malaria, when occurring in temperate climates it is most prevalent in the late summer, or in early autumn. Like malaria, it tends to disappear on the cultivation and drainage and improved sanitation of a country."

In looking over the "Medical and Surgical History of the War of the Rebellion," I have been struck by the apparent accurateness of the observations of the medical officers on the "Pathologic Anatomy and Pathology of Malarial Disease," and the deductions drawn therefrom, measured by the present-day knowledge of the pathologic conditions brought about by the malarial parasite.

The postmortem notes, so far as required for our subject, are as follows:

Stomach.—In 21 of the cases the condition of the stomach is not stated; it was normal in 5 and constricted in 1. In 13 cases a morbid condition was specified thus: In 4 the organ contained a green-grumous or mud-like liquid; in 3 its mucous membrane was congested; in 1 softened; in 1 slate-colored; in 1 thickened, and in 4 ecchymosed.

Intestinal Canal.—In 6 of the 40 cases the intestines were reported healthy; in 9 their condition was not stated. In 13 of the remaining 25 cases the large and small intestines, so far as can be ascertained from the phraseology of the reports, were both affected. Five of the cases were much congested or inflamed, but not ulcerated; in the first-mentioned, for instance, the canal was almost black from the engorged condition of the capillaries, and coagulated blood was found in its interior. In 1 of the 13 cases the mucous membrane was softened, and in 7 ulcerated; in 2 of the latter the duodenum alone was ulcerated, although the canal was generally highly congested; in 1 the ulcerations were reported as slight; in 1 as old; in 1 as large and in 1 as small, and in 1 as associated with a congested condition of the mucous membrane. In 6 of the 25 cases the condition of the small and large intestines is specifically

stated: In 1 both were inflamed, but the patches of Peyer were not diseased; in 1 both were much congested and ecchymosed; in 2 deposits of black pigment were found in the large intestine, while the ileum in 1 was but slightly congested, and in the other merely stained with bile; on the other hand, the patches of Peyer in 1 presented the pigmented appearance, while the rectum was ulcerated; in 1 the mucous membrane of the ileum was softened and thickened, that of the colon congested. In the remaining 6 of the 25 cases the small intestine or ileum only was affected. In 3 it was congested, in one of which the patches of Peyer and the solitary glands were enlarged; in 1 it was ulcerated; in 1 congested to a purple color, which presented a deeper tint in the apices of the solitary glands, although the patches of Peyer were unaffected; in 1 also there was a deeply colored congestion which was especially marked throughout the jejunum.

I might quote the parallel observations of Maillot and of Laveran on the incidence of dysentery in malaria among the French troops; many others have contributed thoughts along this line. Craig, in his book on Estivo-Autumnal Malaria, leans very pronouncedly to the plasmodium as a causative factor in diarrhea and dysentery.

I have seen many, many times cases of dysentery respond promptly to quinin treatment. I am not now possessed of the case notes in these cases, but I remember well to have had a small epidemic of dysentery, each case of which began with the algid form of pernicious malaria. These were not merely of the catarrhal character, but there was "chopped meat" discharges, evidence of some sloughing. All of these cases—about 12 in number—responded to ordinary antiseptic irrigations, gland arousement and quinin in a week or less. Convalescence rapid.

Lastly, malarial parasites have been found not infrequently in the vessels of the mucosa and submucosa in cases of malarial dysentery coming to necropsy.

PATHOLOGY AND CLINICAL HISTORY OF AMEBIC DYSENTERY.

H. F. HARRIS, M.D.
ATLANTA, GA.

PATHOLOGY.

The alterations found in the bodies of those dead of amebic dysentery vary greatly, depending on the time that the disease has existed, the extent of the intestinal lesions, and the complications that have arisen during the course of the malady—some lesions being always found, while others are encountered with greater or less frequency. Among the most common of these is emaciation, which, especially in chronic cases, is often extreme. On opening the abdominal cavity peritonitis is occasionally encountered, which may be very mild, and frequently extends but little beyond that portion of the membrane that surrounds the large gut, but in those cases where perforation has occurred a general suppuration of the entire cavity is found, except it be that adhesions of such a character have previously formed that the contents of the intestine are poured into a closed sac cut off from the general peritoneum; under such circumstances the condition is practically one of abscess formation. Though not invariably the case, the large gut is usually adherent to the coils of the small intestine or other viscera, and it is sometimes united to the walls of the abdomen by newly-formed bands of fibrous tissue; in some cases neighboring portions of the large intestine are united to each other. As a result of these adhesions the colon is often drawn from its normal posi-

tion, and in some cases is greatly distorted. It is generally found that the walls of the gut are so thinned in places that it is only with the greatest care that it can be removed without being torn. On opening the intestine it is found that its coats are much thickened in some situations, and that on its surface there are numerous ulcers. The extent of the ulceration varies greatly in different cases, it being sometimes slight and confined to one portion of the gut, while in other instances the entire mucous surface of the viscus is denuded; it is, however, fortunately true that in fully half of the cases the lesions do not extend upward beyond the beginning of the transverse colon. The ulcers vary in size from those that are microscopic to others that cover a large portion of the mucous surface of the gut; as a rule, however, they are separated from each other by more or less normal mucous membrane. The edges of the ulcers are prominent and project above the general surface. The floors of these lesions are ashen-gray in color, and are almost always covered by a layer of mucus, in which there are particles of undigested food and masses of sloughing tissue; when examined under the microscope this mucus is found also to contain many swollen cells, leucocytes, amebæ and myriads of bacteria. The edges of the ulcers are practically all undermined, and not uncommonly, it is said, fistulous tracts connect them when near each other, though I have never myself been able to demonstrate this. These lesions may be rounded or irregularly shaped, but more commonly they are oblong, and lie transverse to the long axis of the intestine. It is rather curious that these portions of the gut that lie nearest the attachment of the mesocolon are, as a rule, but little affected. The mucosa between the ulcers is usually somewhat reddened, but it may appear entirely normal.

The intestinal lesions of this disease are almost always confined to the large gut, but now and then the process extends up into the small intestine for a short distance.

I saw the appendix involved in one case; the small gut was also affected. It is remarkable that in cases where recovery occurs—even in instances where the disease was severe, and had existed for some time—the large intestine after a short time rarely shows macroscopic evidence of the pronounced ulceration that must have existed.

Our knowledge is very meager as to the exact nature of the finer alterations that occur in the intestine in the early stages of amebic dysentery in the human being, but the changes that take place in the large gut of the dog have been studied by several investigators. The sequence of events that occur in the puppy after having received an injection of material containing living amebæ seems to be briefly as follows:

Collections of amebæ occur on the mucous surface of the intestine, and quickly thereafter the epithelial cells on which these organisms lie exhibit catarrhal changes and drop off from their basement membranes; the blood vessels in the tissues below dilate, and the lymphatics become enormously distended with fluids. There is an increase in the number of lymphoid cells, and many of the large phagocytes of Metchnikoff collect in the part; simultaneously some of the dilated blood vessels burst, and we find as a consequence that there are many red and white cells in the affected area. Following this, the basement membrane on which the desquamated epithelial cells originally lay breaks down and disappears, and the blood that was free in the tissues at once finds its way to the surface and becomes mixed with the mucus and other materials present within the lumen of the gut. Necrotic changes in the tissue surrounding the crypts quickly follow, and these structures fall out, leaving ulcers that extend down to the muscularis mucosæ. The coat just named offers considerable resistance to the downward progress of the ulceration, but it eventually gives way, and the process passes into the submucosa. The tissues composing this

coat in their turn undergo necrotic changes, which extend rapidly down to the circular muscular layer, and laterally in every direction. The walls of these ulcers are not made up of the pre-existing tissues of the part, but are composed of enormous collections of phagocytes of Metchnikoff, and red blood cells, with a smaller number of polymorphonuclear leucocytes and lymphoid cells, and a few plasma cells.

This brings us to the conditions practically always found in the human being dead of amebic dysentery. When we examine the intestine microscopically it is found that all of the characteristic lesions extend into the submucosa, though in rare instances superficial ulcers do occur, which, however, appear not to have any connection with the predominant process, and will therefore not be considered. The mucosa that lies at a short distance from the true ulcers, as a rule, shows astonishingly little change, but as the edges of the lesions are approached the cells lining the crypts often exhibit decided catarrhal change, and the blood vessels in the surrounding tissues are frequently considerably dilated. Amebæ are but seldom found either within the lumina of the glands or in the tissues that lie between them. At the edges of the ulcers the mucous coat generally terminates abruptly, but now and then this layer extends outward over the surface of the ulcer to a greater or less distance; this outgrowth of mucous tissue is composed mainly of deformed and only partially developed crypts that do not become attached to the underlying tissues; these bands are sometimes reflected back over the normal mucosa. In rare instances the process of healing over of the ulcers may be observed; this consists in the epithelial cells growing downward from the edges over the surfaces of the ulcers, and ultimately covering them with a single layer of cells, no new crypts being formed. The floors of the open ulcers are usually formed by one or the other of the muscular coats—generally the inner—though the necrotic process not uncommonly extends

completely through these layers, and we find only the thickened peritoneum interposing between the abdominal cavity and the lumen of the intestine. Irrespective of the depth to which the ulceration has extended the surfaces of the lesions are always made up of partially necrotic tissue, in which there are multitudes of bacteria, many amebæ, and great numbers of cells. In these situations swollen bands of fibrous tissue that appear hyaline and take acid stains intensely are frequently found; they contain no recognizable nuclei. It is noteworthy that the elastic tissue fibrils are quite as well, if not better, preserved in these areas than the fibrous structures; this is exactly opposite to what occurs in the dog under similar circumstances, and may account for the great rapidity with which the tissues in these animals break down. Just beneath the surfaces of the ulcers the pre-existing tissues of the parts are found in varying stages of necrosis depending on the depth, and these structures are separated by accumulations of fluids between them and the presence of a vast number of cells; these consist of polymorphonuclear leucocytes, lymphocytes, plasma cells, connective tissue cells, mast cells and red-blood corpuscles. Amebæ are commonly present in these areas. The blood vessels are for the most part dilated and filled with blood; in many instances the vessels contain thrombi. The vessel walls are frequently necrotic, particularly on the side nearest the ulcers; amebæ are often found within their lumina. The lymph spaces and lymph channels are universally distended with fluids and cells. The epithelial cells that formally lined these vessels become greatly swollen, and almost without exception desquamate, and then lie loosely within the lumina of the vessel; mixed with these cells red blood corpuscles, leucocytes and amebæ are frequently found. Lower down in the walls of the gut these alterations become less pronounced, but the proximity of the pathologic process is usually evidenced by the presence here and there of small collections of

lymphocytes, and great numbers of mast cells are scattered throughout the tissues. Newly formed masses of fibrous tissue are not uncommonly found at a considerable distance from the point of ulceration. Where the necrotic changes have passed through the muscular layers the subperitoneal coat is always greatly thickened, and the tissues composing it exhibit changes similar to those that have been described as occurring in the more superficial portions of the intestinal wall. While it is true that the amebæ, as a rule, are rather behind than in advance of the areas of greatest pathologic change, they are not uncommonly encountered at considerable distances from the edges of the ulcers. Under these circumstances I have been struck with the fact that they are usually acidophilic, and that their nuclei are not colored by basic dyes. Furthermore, numerous bacteria that stain well are found within them, and in many instances the parasites are undergoing disintegration, and the micro-organisms that they contain are being disseminated into the surrounding tissues. The changes that occur in the tissues at the lateral margins of the lesions resemble, on the whole, those that take place at their bases, with the exception only that the structures composing the submucosa are much more susceptible to the action of the poison of this disease than are those that make up the remaining coats of the intestine; this is strikingly shown by the resistance of the muscularis mucosa, which always projects out over the necrotic submucosa, and supports the overlying mucous coat. It is thus seen that the undermined edges of the ulcers in this, as in other intestinal diseases, are due to the power of the muscular coats to resist the poisons that produce the pathologic change. Fibrin is frequently present in the tissues of the submucosa in the neighborhood of the necrotic areas. In the lateral margins of the ulcers amebæ are numerously present, more so than in any other part of the intestinal walls; they lie in the necrotic structures, and somewhat within the tissues

that are more or less normal, but do not penetrate very far as a rule. The changes in the tissues are always in advance of the great body of amebæ, this again clearly showing that if they be in reality the cause of the pathologic process they must necessarily act by producing some poison that is absorbed into the tissues. It is noteworthy that the lymph nodes that are present in the mucosa and submucosa show no marked increase in size, and exhibit, as a rule, but little alteration until the diseased process actually includes them, when they become necrotic and break down just as do the other structures.

Following acute amebic dysentery—and strangely enough appearing to develop only during the first few weeks of the disease—abscess of the liver may occur. These lesions may be situated in any part of the liver, and I do not think from a study of the literature that they are so strictly solitary as they are generally supposed to be, but miliary abscesses, such as occur in pyemia, very rarely take place. Any part of the liver may be involved, but the convex surface of the right lobe is most commonly affected. These abscesses, if not interfered with surgically, may rupture into the intestine or neighboring cavities, and may even open externally; there seems, however, to be a marked predisposition on their part to penetrate through the diaphragm and ultimately break into the lung. When this occurs an abscess of the lung develops, the contents of which eventually discharge through the bronchi. The contents of the liver abscess is made up of rather dark, pus-like material which on microscopic examination is found to consist of a very few cells, a great amount of granular debris, and numerous amebæ. Amebæ are always found in these abscesses—this being even the case in dogs where these lesions follow experimental dysentery. The walls of the liver abscesses may consist merely of disintegrating liver substance, or may be made up where the lesion has existed for some time, of a thick mass of fibrous tissue containing numerous lymphoid and plasma

cells. It is noteworthy that the amebæ never penetrate into the walls of the liver abscesses, but are very frequently found just along the edges. The liver cells in the vicinity of the lesions show a very peculiar degenerative change which consists in the formation of nucleated bodies that bear considerable resemblance to amebæ. Bacteria are usually found in and around the walls of these abscesses. The blood vessels in the area surrounding the lesions usually contain no blood, but are filled with a granular detritus. In some instances areas of rhexis occur around the edges of the abscess.

It appears to me that there can be little doubt that the amebæ reach the liver by means of the portal circulation, though it has been assumed by some that the organisms get into the general peritoneal cavity, find their way to the liver, and burrow into its substance. The fact that amebæ are very frequently found within the blood vessels of the intestinal wall, even when no thrombosis exists, would seem to make it necessary that they would inevitably reach the liver substance.

SYMPTOMATOLOGY.

Amebic dysentery is a disease that frequently sets in acutely, but almost, if not quite, as commonly the beginning is gradual. In the former class of cases there is nothing in the symptoms at first that distinguishes the disease from the ordinary acute dysentery, which is so extremely common in the southern part of the United States in the spring and summer. In these instances the disease begins with diarrhea, quickly followed by stools of a dysenteric character; the patient complains of pain and tenesmus in the abdominal region, and there is at the same time a decided quickening of the pulse and some fever. In the course of a few days or weeks the severity of the symptoms usually somewhat subsides, but in exceptional instances the patient slowly grows worse, and death occurs in from two to three weeks. In those cases where improvement follows the acute onset the disease seems always to become chronic,

and it is in this stage that the patient usually consults the physician. Where the disease begins gradually the sufferer is rarely able to state with definiteness the time when his trouble began; he remembers only that about a certain time he began to note that his bowels would be loose for a day or so, this being followed by constipation. There were a number of such periods of alternating constipation and looseness of the bowels, but it was observed that the period between the diarrheal attacks became less and less, until finally a condition was reached where there was several movements of the bowels in twenty-four hours. About this time the patient usually begins to observe that the stools contain some blood, and he has slight attacks of pain and tenesmus at the time that the bowels act. At the end of a few weeks the symptoms in every way resemble those that are encountered at an equal period of time from the onset in those instances where the disease began acutely. It is in this stage, after the malady becomes chronic, that it generally comes to the notice of the clinician, as in the class of persons who suffer from this affection a physician is very rarely called in merely for an acute attack of bowel trouble. After the disease becomes chronic it runs an extremely irregular course, sometimes getting well without any apparent cause in a comparatively short period of time, while in other instances it exists for many years. Some time ago Dr. Reginald Fitz of Boston wrote me that he had under his care a patient who had suffered with this malady since the Civil War, and I have myself seen many instances where the disease continued four, six and even eight years. Though somewhat arbitrary, for convenience sake, the affection may be clinically divided into several types, it being understood, however, that either of these may pass into the other at any time, and without any apparent cause:

1. *The Very Mild Form.*—The general health remains fairly good, there is no fever or acceleration of the pulse

worthy of mention, and the appetite is good. The number of stools vary from two to six in twenty-four hours. This is the form almost always seen in children.

2. *The Moderately Severe Form.*—In this type of the disease the general health is never good, the appetite is, as a rule, poor, and there is considerable loss of flesh. The pulse is increased in frequency and the temperature goes up toward evening. The number of stools vary from six to twenty in twenty-four hours.

3. *The Very Severe Form.*—In this form of the disease there is great emaciation and loss of appetite, with sometimes nausea and vomiting. The temperature is irregular, but in the afternoon is usually above normal, and the pulse is weak and decidedly accelerated. The extremities are cold, and profuse sweating is quite common. Hiccough is frequent. The stools are watery, and consist very largely of blood and mucus, in which sloughs are sometimes found. The number of stools vary from twenty to fifty in twenty-four hours. It is very remarkable how patients may remain in a condition of this kind for days, and even weeks, and finally recover. It seems to be a peculiarity of all intestinal affections that death may be very closely approached without a fatal termination actually occurring.

There are very remarkable fluctuations in the severity of the symptoms in all forms of the malady, it being not an uncommon observation that the disease passes quickly from the mildest type to the most severe, and vice versa. While we can not always account for an increase in the severity of the clinical course of this affection, it is true that bodily exertion is often followed by a decided exacerbation in the symptoms, and the taking of certain articles of food that disagree with the sufferer frequently occasion distressing results; alcoholics seem to be especially badly borne—the taking of beer being particularly injurious.

Among the symptoms that are occasionally encountered may be mentioned a constant aching in the back that is not relieved by the recumbent posture, a tear-

ing sensation in the region of the colon, great uneasiness in the stomach, and an urgent desire to defecate immediately following the taking of food and edema of the face and feet. In the mild form of the disease the urine is practically normal, but in the severer types albumin is occasionally present. I have examined repeatedly the urine of some of my worst cases for peptone by the method of Hoffmeister, with negative results. In the severer forms of the disease the chlorids are often diminished, and in the worst cases may be totally absent. Their reappearance after having been absent from the urine is a most favorable indication. In the milder forms of the disease the stools may appear almost normal in every way, but a careful inspection will usually reveal the presence of small particles of bloody mucus upon the surfaces of the fecal masses. In the severer types of the malady the movements contain considerable quantities of mucus and blood, and are quite soft in consistency. In the worst forms the fecal discharges consist almost entirely of blood and mucus; stools of this character, occurring in a chronic intestinal affection, I consider entirely characteristic, and even in the absence of a microscope I should feel but little hesitation in making a diagnosis of amebic dysentery. In some cases just before death the quantity of mucus becomes decidedly lessened, the stools consisting of an extremely thin, dark, blood-stained fluid. The feces are, as a rule, alkaline in reaction, but are occasionally slightly acid in mild cases. On microscopic examination the stools are found to consist of partially digested food, much mucus and blood, pus, large epithelioid cells, multitudes of bacteria, and varying numbers of amebæ. Cercomonas are sometimes encountered.

The most important of the complications of amebic dysentery is liver abscess; out of ninety-five cases this has occurred fifteen times—once in a boy. As has been before stated, this complication always arises during the acute period of the disease, there being no instance with

which I am acquainted in which it occurred later. It appears to be more common in the northern portions of our country than in the south, as I myself have seen this in only two instances out of fifty-two cases. It is, however, quite likely that the proportion is greater than this, even here, as almost all of my cases were seen after the disease had become chronic. The symptoms of liver abscess are by no means always characteristic, there being many instances recorded where the disease lay latent for a considerable period of time without it being suspected. The usual symptoms are a throbbing or aching pain in the region of the liver, which may be reflected to the right shoulder, chills and fever of an irregular character, and rapidity of the pulse; the appetite fails, the tongue becomes coated, the patient rapidly loses flesh, and jaundice is occasionally encountered. On physical examination the liver is found to be enlarged; a fullness or marked protuberance sometimes develops at the lower margin of the ribs.

When the liver abscess opens into the lung, as it most frequently does, abscess of this organ develops. The symptoms of this condition are the usual signs of consolidation in the lung, with cough and the expectoration of a reddish-brown fluid, which on examination with the microscope is found to contain amebæ. This complication is rare, it having occurred in only three instances out of ninety-five cases of amebic dysentery.

Perforation of the intestine with peritonitis is an occasional complication of this disease; the symptoms are those that are usually observed in cases of this kind. It has appeared to me that the inflammatory phenomena are not so marked as in the septic peritonitis that follows operations.

Appendicitis has been observed in one case. The symptoms would naturally be those commonly observed where this organ becomes diseased, but in the instance to which I refer the clinical course was so mild that the true condition of affairs was not suspected during life.

A STUDY OF THE CASES OF AMEBIC DYSENTERY OCCURRING AT THE JOHNS HOPKINS HOSPITAL.

THOMAS B. FUTCHER, M.B. (TOR.)
Associate Professor of Medicine, The Johns Hopkins University.
BALTIMORE.

(From Professor Osler's Clinic, The Johns Hopkins Hospital.)

It is not the intention in this paper to attempt a complete historic account of amebic dysentery. I wish merely to recall the fact that it was Lösch,[1] of St. Petersburg, who in 1875 was the first to give an accurate description of amebæ in the stools of a dysenteric patient. Although Lambl and Cunningham had previously described amebæ in the stools of infants and cholera patients, it remained for Lösch to associate the amebæ with dysentery in the relation of cause to effect. It was also Lösch who gave to the organism the name *Amœba coli,* a name that is still in very general use. Kruse and Pasquale advocate the adoption of the term *Amœba dysenteriæ,* first suggested by Councilman and Lafleur, to designate the pathogenic ameba, reserving the name *Amœba coli* (Lösch) for the non-pathogenic ameba occasionally found in the normal healthy intestine. In America, amebæ were first found by Dr. Osler,[2] who observed them in 1890 in the pus of a liver abscess originating in Panama and operated on by Dr. Tiffany, of Baltimore. I purpose attempting to give as concise an account of the disease as possible,

1. Lösch: Virchow's Archiv, Bd. lxv, 1875.
2. Osler: Johns Hopkins Hospital Bulletin, vol. i, 1890.

basing the description largely on our observation of the disease and its complications as seen at the Johns Hopkins Hospital.

RELATIVE FREQUENCY.

From May 15, 1889, the date of the opening of the Johns Hopkins Hospital, to May 1, 1903, or for a period of nearly fourteen years, there have been in all departments 120 cases of amebic infection. This was out of a total of 42,614 admissions to the institution. Of the 120 cases, 118 had been admitted to the medical wards. The total number of medical patients during this period was 15,817, so that the medical amebic cases constituted 0.74 per cent. of the total medical admissions. Of the two remaining cases, which were admitted to Prof. Halsted's surgical wards without having been medical cases before or after, one had an amebic abscess of the liver and the other an amebic abscess of the floor of the mouth. It may be safely said that these 120 cases represent pretty accurately practically all the cases of amebic infection that have been admitted to the hospital. It is doubtful whether very many have escaped observation. Since Dr. Osler's first observation, in 1890, and the appearance of Councilman and Lafleur's[3] classical monograph on the subject, in 1891, careful examination of the stools and contents of hepatic abscesses have been made in all suspicious cases. Excluding the case of abscess of the lower jaw containing amebæ, the remaining 119 cases were made up of amebic dysentery or amebic hepatic abscess cases. Only cases in which the histories positively state that amebæ were found are included in the series. Three cases in which a diagnosis of amebic dysentery was made, but in which there is no record in the history of amebæ having been found in the stools, have been omitted. These were in all likelihood actual amebic infections, however, the failure to note the presence of amebæ being an oversight. So

3. Councilman and Lafleur: Johns Hopkins Hospital Reports, vol. ii, 1891.

also in connection with hepatic abscesses, only those in which there is a definite statement that amebæ were found in the contents of the abscess or in sections of the abscess wall have been included. Those in which no amebæ were found, but which proved sterile on bacteriologic examination (a fact pointing rather strongly toward an amebic origin), have been omitted.

ETIOLOGY.

After the discovery of *Bacillus dysenteriæ* (Shiga) as a cause of epidemic and sporadic dysentery, there was for some time considerable doubt expressed as to whether the *Amœba dysenteriæ* played such an important rôle in the causation of amebic dysentery. It was thought that possibly the intestinal ulceration was first started by the Shiga bacillus, and that the ameba was a later invader, and played a rather secondary rôle. Those who have had most experience with the dysentery bacillus, including Flexner among others, state that there is unquestionably a form of dysentery which is quite independent of that produced by that organism and actually due to the inroads of the *Amœba dysenteriæ.* We can again, therefore, use the term amebic dysentery with full confidence. The pathogenic agent is unquestionably the *Amœba dysenteriæ.*

This parasite is placed by Leuckart in the class *Rhizopoda* of the *Protozoa.* It is a unicellular organism, varying in size between 8 and 37 microns, according to Braun,[4] but usually between 12 and 26 microns, the average diameter being about 20 microns. Whereas in different cases the amebæ may vary greatly in size, in the same case they are usually of a fairly uniform dimension. The body of the ameba consists of two portions: the inner granular portion is called the endosarc or entoplasm; the outer, clearer, hyaline portion is termed the ectosarc or ectoplasm. The division into these two parts is not clearly made out in the resting stage, but be-

4. Braun: Die Thierischen Parasiten des Menchen, Zweite Auflage, p. 44.

comes very distinct in the actively motile organisms. The greater part of the body of the cell is comprised of the finely or coarsely granular endosarc. This portion often contains several vacuoles of varying size. A nucleus can occasionally be made out in the fresh unstained ameba; more distinctly in the stained preparations. It averages about 6 microns in size, and is situated in the endosarc, near its border. A nucleolus can be recognized in the stained specimens. The ameba frequently contains foreign bodies. Red blood cells constitute one of the commonest inclusions. The number of red cells contained may reach as high as fifteen or twenty. Included red cells are apparently more numerout in acute cases. Micrococci, bacilli and granular debris are not infrequently seen within the body of the cell.

Probably the most striking thing about the *Amœba dysenteriæ* is its motility. This may present itself in two forms, either a change in shape or a change in place. The change in form is also of two kinds. A pseudopodium, consisting of a process from the hyaline ectosarc, may be seen to be protruded, and in a few minutes attracted into the body of the cell again. A more remarkable change in form is occasionally observed. This consists in the sudden throwing out of a process of the ectosarc, which instantaneously partially or completely encircles and envelopes the cell. The movements of locomotion are most interesting to watch. They seem almost purposive in their character. A hyaline process is thrown out from one border of the parasite, and the granular endosarc, with its inclusions, rapidly flows into the process. By the continual throwing out of a process from the same side of the cell an ameba in a very few seconds will cross the field of the microscope. In some of these very actively motile parasites it is almost impossible to differentiate ectosarc and endosarc, the ameba changing its place by a rapid rolling or flowing motion. In a general way, it may be stated that the more acute

the course the more rapid seem to be the movements of the amebæ. When a satisfactory portion of mucus has been obtained, almost the entire field with the oil immersion lens may be seen to be comprised of very actively motile organisms. Cold tends to retard the activity of the parasites, although they will remain fairly active, often for hours, at the room temperature. When the temperature falls below 75 F. the ameboid movements cease, but they may be again started by gently raising the heat to the body temperature by carefully warming the slide over a spirit lamp. With a warm stage, the amebæ will retain their motility almost indefinitely. The activity of the ameba is immediately arrested by a solution of quinin of the strength of 1 to 5000. During the process of death of the organism vacuolization of the cell occurs.

It is not known definitely how the ameba reproduces itself. By analogy, however, one is lead to assume that this takes place by fission or sporulation. The ameba is stained by various basic anilin dyes. The staining methods, which will not be given here, are not perfectly satisfactory, although there is no difficulty in recognizing the organism in stained sections of an intestinal ulcer or wall of an hepatic abscess. Mallory and Wright[5] advocate thionin as a differential stain. Their method aids very materially in differentiating amebæ in sections from mastzellen, with which they are liable to be mistaken. With their thionin stain the nuclei of the amebæ and the granules of the mastzellen are stained brownish-red; the nuclei of the mastzellen and of all other cells are stained blue.

Resistant forms of amebæ, similar to the gamete forms of the malarial parasite, have been noted by Cunningham, Grassi and Calandruccio, and by Quincke. These "encysted amebæ" are believed by these observers to be necessary, under certain conditions, for the transmission of the disease from one person to another.

5. Mallory and Wright: Pathological Technique, p. 293.

The ameba is very difficult to cultivate. Kartulis used straw-infusion, and claimed to have been able to grow the parasite. Objections were later made to his technic, as it was believed that his positive results may have been due to accidental contamination. Celli and Fiocca[6] succeeded in obtaining pure cultures of the ameba. They describe two phases in the life cycle—the ameba form and the cyst form—and they state that the amebæ multiply by division.

(*a*) TYPES OF AMEBA FOUND IN THE STOOLS.

It is now well recognized that amebæ may occur in the stools of healthy persons or in the discharges of patients suffering from other diseases, without their having in these cases any pathogenic action. Thus they have been found in cases of chronic diarrhea, intestinal tuberculosis, typhoid fever, hemorrhoids, and other affections.

As a result of careful study of the amebæ found in the feces, supported by inoculation experiments, Quincke and Roos[7] have been led to the conclusion that there are three different varieties of amebæ in the stools of human individuals. The names which they give these different varieties, with their pathogenic actions, are as follows:

1. *Amœba intestini vulgaris,* 40 microns in diameter, coarsely granular, and not pathogenic to either human beings or cats.

2. *Amœba coli mitis,* the same size, pathogenic for man, but not for cats.

3. *Amœba coli* (Lösch) or *Amœba coli felis,* up to 25 microns in diameter; pathogenic for both man and cats, and producing dysentery in both.

It will be seen that the second variety, the *Amœba coli mitis,* which is pathogenic for man only, is much

6. Celli and Fiocca: Centralblatt f. Bakteriologie, vol. xv.
7. Quincke and Roos: Berl. klin. Wochenschrift, Bd. xxx, 1893, p. 1089.

larger than the third variety, the ordinary *Amœba coli* (Lösch).

In our series of 119 cases of dysentery we have no positive evidence that we were dealing with any other than the usual *Amœba dysenteriæ*. In one or two cases, owing to the average diameter of the parasite found being rather smaller than that of the ordinary ameba, it was thought that we might be dealing with a special type of the parasite.

(*b*) DISTRIBUTION OF THE AMEBÆ IN THE HUMAN BODY.

The usual habitat for the *Amœba dysenteriæ* is in the large intestine. Where complications follow, it may be found in other situations. Thus it occurs in the contents of liver abscesses, in the sputum of patients with hepato-pulmonary abscess, and in the pleural and peritoneal cavities when a liver or pulmonary abscess breaks into one or other of these. Other situations in which amebæ have been found are as follows:

1. In the buccal cavity—the so-called *Amœba gingivalis* of Gros and *Amœba buccalis* of Sternberg. These forms were found in the tartar scraped from the teeth, and are regarded as being identical with the *Amœba dysenteriæ*.

2. In the urogenital tract. In 1889, Baelz described, under the name of *Amœba urogenitalis*, large, actively motile amebæ in the urine and vagina of a young woman, 23 years of age, who died of pulmonary tuberculosis. Other similar observations were reported.

3. In pieces of necrotic bone. Kartulis observed amebæ in the pus from an area of necrosed bone involving the lower jaw of an Arab, aged 48.

4. In an abscess in the floor of the mouth. This observation was reported by Flexner.[8] The case occurred in Dr. Halsted's wards in the Johns Hopkins Hospital, and, as already stated, is the only one in this series of 120 cases that was not an amebic dysentery

8. Flexner: Johns Hopkins Hospital Bulletin, vol. iii, 1892, p. 105.

or amebic hepatic abscess case. The patient was a male, white, aged 62 years. The amebæ were obtained from the contents of the abscess at operation. Flexner believed that this form of the organism was closely allied to, if not identical with, the *Amœba dysenteriæ.*

(*c*) SOURCE OF THE AMEBÆ AND MODE OF INFECTION.

Very little is known regarding the source of the ameba and the mode of infection. We are still absolutely unfamiliar with the life history of the parasite outside the body. Inoculation experiments by way of the mouth, with the possible exception of those of Quincke and Roos, with the so-called encysted forms, have been entirely negative. It is highly probable that infection takes place by way of the gastro-intestinal tract, and that amebic dysentery is contracted in much the same way that typhoid fever is.

GENERAL ETIOLOGIC FACTORS.

(*a*) *Geographical Source of Infection in the Johns Hopkins Series.*—The term "tropical dysentery," for many years used almost synonymously for "amebic dysentery," has long been recognized as a misnomer. Amebic dysentery is not confined to the tropics, but often originates in the subtropical, even in the higher temperate zones. Sporadic cases occur in New England, and it will be remembered that last year Dr. Dock[9] reported before this Section a case which originated in Michigan. As the analysis of our cases shows, the disease occurs very frequently in Maryland and the bordering states. Of the 119 cases of amebic dysentery, the disease was apparently contracted in Baltimore in 83, and in the state of Maryland, outside of Baltimore, in 13 cases, or a total of 95 cases contracted within the state of Maryland; 2 in the District of Columbia; 8 in Virginia; 1 in West Virginia; 1 in North Carolina; 1 in Tennessee; 1 in Georgia; 1 in Alabama; 2 in Florida; 1 in Cuba; 1 in Brazil; 4 of

9. Dock: THE JOURNAL of the American Medical Association, 1902.

doubtful source, the patients being sailors or travelers, and it being impossible to determine where the dysentery was contracted. It will thus be seen that the disease is very frequently contracted in latitudes as far north as Baltimore.

(*b*) *Age.*—The incidence of the disease in the various decades is shown by the following figures: There were 11 cases in the first decade; 8 in the second; 35 in the third; 29 in the fourth; 20 in the fifth; 11 in the sixth; 4 in the seventh; 1 in the eighth. It will be seen that the largest number of cases occurred in the third decade, 35 cases occurring betwen the ages of 21 and 30. The youngest patient was a male child 2 years 8 months old, and the oldest was a man 71 years of age. The analysis of the series regarding the question of age is of interest in showing the rather frequent occurrence of amebic dysentery in very young children. As stated, there were 11 cases, or 9.4 per cent. of the total, in the first decade, 6 occurring between the ages of 1 and 5, and 5 between 6 and 10 years. It should teach us to keep in mind the possibility that a chronic dysentery or diarrhea in young children may be of amebic origin. This fact was emphasized by Amberg[10] in a paper in which he reported five of the cases of amebic dysentery in children from Dr. Osler's wards.

(*c*) *Sex.*—There is much disparity in the relative prevalence of the disease in the two sexes, as illustrated by this series. There were 108 males and 11 females. It is possible that the out-door life of men render them more liable to infection, by drinking water containing amebæ or by conveying the parasite to their mouths on dirty hands.

(*d*) *Race.*—The series was composed of 107 white and 12 colored patients. Considering the fact that the proportion of whites to blacks in the hospital is about in the ratio of 7 to 1, it would appear that whites

10. Amberg: Johns Hopkins Hospital Bulletin, vol. xii, December, 1901.

are a trifle more frequently attacked, as the proportion of whites to blacks affected was approximately 9 to 1.

(*e*) *Occupation.*—There was nothing in the consideration of the occupation of the patients to throw any special light on the source of infection.

CLINICAL TYPES.

Three groups of amebic dysentery may be made according to the severity of the intestinal symptoms. The cases may be classified as follows: 1. Dysentery of moderate intensity. 2. Grave or gangrenous dysentery. 3. Chronic dysentery. Owing to lack of time, and in order to avoid repetition, these three types will not be described separately. Moreover, the classification is a more or less arbitrary one, and cases may conform to one or other type at different periods. Thus a case of dysentery of moderate intensity may, and often does, become chronic, while a chronic case may take on acute manifestations and assume a gangrenous type.

SYMPTOMATOLOGY.

In many cases the onset is abrupt; in others it is more gradual. It is most acute in its onset in the gangrenous type, which is seen less frequently in the latitude of Baltimore than in the tropics. In the majority of cases the patient is rather suddenly seized with an attack of diarrhea. In a few hours, or a day or two, the dejections assume a dysenteric character, blood and mucus constituting the bulk of the movement. In the severest cases there may be as many as twenty or thirty such movements in the twenty-four hours. In the severe, acute gangrenous cases, large portions of sloughing mucous membrane may be passed. The appetite is impaired, and there may be nausea and vomiting in the initial stages. There is usually fever at the onset, the temperature ranging often betwen 100 and 102 F. When the cases become chronic the temperature may become subnormal. Frequently there is considerable abdominal pain and tenderness in the acute stages. The pain is

most marked over the lower part of the abdomen, although it may be generally distributed. It is intermittent and of a colicky character, and usually is worse preceding the movements. In the chronic cases there may be no pain, excepting during the acute exacerbations. Tenesmus may occur, but it is rather the exception. This is explained by the fact that the lesions are usually most marked in the cecum and ascending colon, the rectum in the majority of cases being uninvolved, or only to a slight extent. The tenesmus in bacillary dysentery, on the other hand, occurs much more frequently, and is often very severe, owing to the rectum being usually much more extensively involved. The degree of emaciation in some cases may be extreme, and is only surpassed by that observed in carcinoma of the esophagus. It is rather surprising, however, how little the general health of the hospital cases is affected in many instances, the color and general nutrition being extremely good. The majority of our cases were of moderate intensity, and in these the symptoms soon subsided under proper treatment. In a considerable percentage of cases the disease is subacute from the start, and gradually passes over into the chronic form. These cases are characterized by alternate attacks of diarrhea and constipation. In the periods of exacerbation the symptoms are rarely so severe as in the acute cases, and the diarrhea is usually readily amenable to treatment. These are the cases which are often so difficult to actually cure, and are the ones that so frequently apply for readmission to the hospital. Of our 119 cases, 19 were readmitted one or more times, with a relapse of the dysenteric symptoms. Of these, 12 were readmitted once; 14 twice; 1 three times, and 2 four times. One patient was admitted to the hospital five times in the short period of nine months; another was admitted five times during a period of one year and seven months. During the exacerbation there is pain, frequent passage of mucus and blood, and usually

slight elevation of temperature. The patients, as a rule, do not feel very ill. Nutrition remains fairly good, and the marked emaciation usually observed in these chronic cases in the tropics was practically never seen in our hospital series. The numerous evacuations in these cases very frequently give way to constipation in a few days, when the patients are kept in bed and placed on bowel irrigations.

CHARACTER OF THE STOOLS.

As stated, in the earliest days of the attack the movements are very frequent in the severest cases—as many as twenty or thirty movements occurring in the twenty-four hours. These gradually diminish in number until, in fatal cases, there may be only three or four movements daily for the last few days preceding death. This diminution is in large part due to the gradual loss of expulsive power, brought about by the destruction of the muscular coat and the general edema of the bowel.

The earliest stools are small in amount; they consist, after the expulsion of the fecal contents, almost entirely of clear or slightly turbid and very viscid masses of blood-stained mucus, mixed with more or less bright blood. With the progress of the ulceration the stools become more copious, watery, and less homogeneous, and there is less blood. They contain, also, small, opaque or translucent, gelatinous, grayish-yellow masses, varying in size from a pin's head to a split-pea; these are fragments of necrotic tissue from the bases of the ulcers, and they contain amebæ. Where extensive and rapid sloughing occurs, the stools become greenish, grayish, reddish brown, or a mixture of these colors; their consistence is more watery, and they have a penetrating and horribly offensive odor. Not infrequently they contain large sloughs in the form of tough, stringy masses of a grayish or brownish color.

In cases of moderate severity with sudden onset the stools resemble those of the early stages of the grave form. The amount of blood and mucus is liable to be

less. In the chronic form the stools are homogeneous, for the most part watery or like thin gruel, excepting during the periods of constipation, and of an earthy, yellowish-brown color. They contain fewer flakes of mucus and seldom any blood or necrotic fragments.

Microscopic Examination of the Bloody Mucus.—In the acute cases, besides the amebæ, whose characteristics have been already described, large numbers of red blood cells, few leucocytes, and a variable number of degenerated intestinal epithelial cells are seen. In a great many of our cases eosinophiles have been present in large numbers. They were found in the stools of all the five children with amebic dysentery reported from Dr. Osler's clinic by Amberg.[10] Charcot-Leyden crystals in rather small numbers are not infrequently present. They usually are most numerous where eosinophiles are present in large numbers. They occurred in four of Amberg's five cases. The stools also contain an innumerable variety of the ordinary intestinal bacteria.

Parasites Other Than the Ameba.—These are not infrequently associated with this organism in the stools. Ciliated infusoria—the *Trichomonas intestinalis* and *Cercomonas intestinalis*—are comparatively frequent associates. When they occur they are usually present in large numbers. One case in this series was of extraordinary interest in that, with the amebæ in the stools, there was also an intestinal infection with the *Strongyloides intestinalis.* This case was reported by Thayer, and is of particular interest in that it was the first case of so-called Cochin-China diarrhea or infection with the *Strongyloides intestinalis* observed in this country. The stools of this patient also contained large numbers of cercomonadids and trichomonadids.

CONDITION OF THE BLOOD.

Anemia of a mild secondary type usually exists, the reduction in the hemoglobin being more marked than that of the red corpuscles. At first thought it would seem natural to expect that with a chronic disease of

this kind the red cells would be quite markedly reduced. This, however, was not found to be the case, the red cells, in fact, being nearly normal. In analyzing the blood counts in our records, I have for purposes of comparison kept the cases with amebic abscesses separate from the uncomplicated amebic dysentery cases. In the case of the red counts and the hemoglobin estimations, the averages were made from the first blood reports recorded in the histories after admission. In order to ascertain whether the leucocyte count was of any material value in determining the presence or not of an hepatic abscess, I have selected the highest leucocyte counts in both groups during their entire stay in the hospital. In many of the early cases no blood counts were made, while in the more recent cases in many instances a complete blood count was not made. This accounts for the averages of the red cells, leucocytes and hemoglobin being made from varying numbers of cases.

1. *Cases with Uncomplicated Amebic Dysentery.*—The average of the red cells in 38 cases was 4,802,000 per c.mm.; the average number of leucocytes in 43 cases was 10,600 per c.m.; and the average hemoglobin estimation in 36 cases was 63 per cent. In five cases the red cells were 6,000,000 per c.mm. or over. The comparatively high average of the red cells and the high count in these five cases are probably explained by a concentration of the blood resulting from the frequent bowel movements. It will be further seen that there was a considerable reduction in the hemoglobin and only a very slight leucocytosis. The average blood count then in the group was, red cells, 4,802,000; leucocytes, 10,600, and hemoglobin 63 per cent.

2. *Cases Complicated by Amebic Abscess of the Liver.*—The average number of red cells in 15 cases was 4,250,000; the average number of leucocytes in 15 cases was 18,350; and the average hemoglobin estimation in 13 cases was 66 per cent. The average blood count in this

group was as follows: red cells, 4,250,000; leucocytes, 18,350, and hemoglobin 66 per cent. It will be seen that the red cells were only 750,000 lower than in the previous group. The leucocytosis was not a high one. The average was 7,750 higher than in the uncomplicated dysentery cases. There were two cases in which the highest count was under 10,000. The highest leucocytosis of all was 53,000.

Some observers hold that there is a great increase in the leucocytes in cases of amebic hepatic abscess and emphasize its importance in the diagnosis of such cases. Few, however, give counts supporting their statements. Although our series shows that the average leucocyte count in the amebic abscess cases is considerably above that in the uncomplicated cases, yet it is doubtful whether the leucocyte count is of any great value as a means of diagnosis. For instance, in the uncomplicated series there were ten cases in which the leucocyte count was above the average in the abscess cases. In two of these the counts were 40,000 and 47,000 respectively.

The average differential count of the leucocytes showed a relative increase in the polymorphonuclears. Considering the rather large number of eosinophiles seen in the stools in certain cases, it was thought that there might be some change in the relative percentage of these cells in the blood. With the object of determining whether there was such a change or not, Dr. McCrae made differential counts in a number of cases, but found the eosinophilic cells present in practically normal percentages.

COMPLICATIONS.

1. *Hepatic Abscess and Hepato-Pulmonary Abscess.*—Abscess of the liver is the most frequent and most serious of the complications of amebic dysentery. Of the 119 cases of amebic dysentery, 27, or 22.6 per cent., developed hepatic abscess. It is impossible to draw an accurate comparison between this percentage and the frequency of this complication in the extensive statis-

tics of British and French physicians dealing with dysentery in India, owing to the lack of mention of the etiologic factor in the latter. In a hospital series, the percentage of hepatic abscess cases would probably be higher than would be the actual percentage outside hospitals, owing to the fact that the severity of the symptoms from this complication would cause the victim to seek admission, whereas the patient with dysentery alone would not be so likely to enter a hospital. There were 24 cases in males and 3 in females. According to decades, the cases occurred as follows: second, none; third, 10; fourth, 5; fifth, 7; sixth, 3, and seventh, 2. It will be seen that the largest number, 10, or 35.7 per cent. of the total cases, occurred between 21 and 30 years. The complication was also most frequent in this decade in Waring's more extensive statistics. Whether there were any special determining factors predisposing the patients to the development of this complication could not be ascertained. In one case direct traumatism in the hepatic region seemed a factor. The Anglo-Indian authorities lay great stress on the predisposing influences, particularly of alcohol, but also of condiments and high temperature.

Relationship to Onset of Dysentery.—The series was analyzed for the purpose of determining the length of time the dysentery had lasted before the symptoms of hepatic abscess manifested themselves. The result was as follows: In 3 cases there was no statement as to how long the dysentery had lasted. In 5, the intestinal manifestations had been so unimportant as to lead the patient to state on inquiry that he had not had diarrhea. Definite data are given in 19 other cases. In eight cases the dysentery had lasted one month or less. In one of these the dysentery and abscess symptoms had come on simultaneously, and in three others the dysentery had lasted only two weeks; in 1 case the dysentery had lasted two months; in 1, three months; in 2, four months; in 1, five months; in 2, six months; in 1, seven months;

in 1, eight months; in 1, one year, and in 1, six years. The statement is sometimes made that hepatic abscess only develops in the very acute stages. The above analysis amply proves the contrary. It shows, however, that the largest number did develop within the first month after the onset of the dysentery. It must be emphasized that in one case the intestinal symptoms had lasted one year and in another case the remarkably long period of six years. Probably the most interesting fact of all is that in 5 cases the intestinal disturbance had not been sufficient to attract the patient's attention. In 22 of our 27 cases of hepatic abscess there was clinically or anatomically the existence of amebic dysentery, although in two of these cases there were only the scars of old ulcers. In two of the remaining five cases the intestine seemed normal at autopsy. This is an interesting fact, as it shows that the ulceration may be so slight as to cause no evidence after healing, and yet hepatic abscess may result in such cases. The other three cases were among those that recovered, and no history of dysentery had been obtained, amebæ being present, however.

Number of Abscesses.—The belief, formerly held, that amebic hepatic abscess is nearly always single, is no longer tenable. There may be only one large abscess, but not infrequently there are two or three fairly large abscesses, while, on the other hand, there may be thousands of minute abscesses, some almost indistinguishable to the naked eye. These minute abscesses have also been shown to contain amebæ. In 8 of the cases the abscesses were multiple. The most frequent situation for the single large abscess or for the multiple large abscesses is the upper and posterior part of the right lobe of the liver under the diaphragm. Autopsies were obtained in 22 of the 27 fatal cases of amebic dysentery. Eighteen of these cases were found to have hepatic abscesses. In 10 there was a single large abscess in the upper part of the right lobe.

Spontaneous Rupture of Abscess.—Amebic abscess of the liver may rupture spontaneously. In our series of 27 cases the abscess ruptured into the surrounding viscera or tissues as follows: Through the diaphragm, producing abscesses in the right lung or discharging themselves through the bronchi in 9 cases; into the right pleural cavity, causing an empyema in 3 cases—in one of these a pyo-pneumothorax was recognized clinically; into the duodenum in 1 case; into the inferior vena cava in 3 cases. In one case the abscess extended into the retroperitoneal tissues, involving the muscles of the back and the psoas muscle. In another an abscess in the lower part of the right lobe of the liver invaded the right kidney. This explained a hematuria that the patient had previous to admission. One of the cases was remarkable in that a large abscess in the upper part of the right lobe had entirely healed and had been replaced by a mass of dense fibrous tissue measuring about 3 cm. in diameter. This abscess had previously ruptured through the diaphragm into the lung, and the patient died from the effects of a large abscess in the lower lobe of the right lung, in which amebæ were found. The opening in the diaphragm was found entirely closed at autopsy. Seven of the abscesses were intact at the time of the autopsy. The series shows, what all statistics have, that the rupture is most frequently through the diaphragm into the lower lobe of the right lung with the establishment of an hepato-pulmonary abscess. The relatively large number of ruptures (three) into the inferior vena cava is of extreme interest. In one case, the inferior vena cava, beginning at a point just above the rupture, contained an organized, decolorized, firm thrombus, which extended upward and projected into the right auricle, which it nearly filled. In addition to the situations above noted where rupture may take place, the abscess may open into the pericardium, the stomach, bladder, colon, or small intestine, peritoneum, or through the skin in the right hypochondrium.

Symptoms of Hepatic Abscess.—The symptoms may be insidious in their onset, or they may be ushered in fairly abruptly. Pain is one of the earliest symptoms. At first it is usually dull and aching, later becoming lancinating and tearing in character. The former is due probably to stretching of the liver, while the latter is attributable to involvement of the peritoneum or pleura. The situation of the pain varies greatly. It may be in the epigastrium, right hypochondriac, lower right axillary, or scapular region. It is quite surprising the large number of our cases that have complained of pain in the region of the right shoulder-joint. This is a reflex pain through the phrenic, which receives large twigs from the fourth cervical. The fever which develops is at first usually of a continuous type; later it becomes remittent, and still later of an intermittent, septic type. At this stage chills are common, and the symptoms may simulate malaria. Profuse sweating is the rule. The pulse ranges between 100 and 140, and there is a certain amount of dyspnea, especially with any pleural or pulmonary involvement. There may be nausea and vomiting. Emaciation may be very marked. The patients have a sallow icteroid tint, and the conjunctivæ have a yellowish hue. The French think that there is something almost pathognomonic in the facial appearance of these cases. Marked jaundice is quite rare. As shown above, there is usually a moderate leucocytosis, the average in our cases being 18,300 per c.mm.

Physical examination may show some fullness in the epigastrium or right hypochondrium. There may be tenderness over the hepatic region, and if the abscess has attained considerable size the edge of the liver will be palpable. If the abscess be situated on the lower aspect of the right lobe or in the left lobe, its presence is more likely to be suspected early then when situated elsewhere. Percussion, when the abscess is of considerable dimensions, will nearly always reveal an increase in the area of hepatic flatness. Owing to the fact that

the large abscesses are most frequently situated in the upper and posterior part of the right lobe, it will usually be found that the hepatic flatness will reach much higher than normal in the axillary line, and extending as high as, or higher than, the angle of the scapula. Auscultation may reveal nothing unusual. There may be an audible peritoneal rub over the liver, or, if the inflammatory trouble has extended through the diaphragm, there may be a pleuritic friction rub.

If the extension of the abscess upward be gradual, adhesions may take place betwen the visceral and diaphragmatic pleura, and the abscess may open directly into the lung, with the establishment of a hepato-pulmonary abscess. Rupture of the hepatic abscess into the lower lobe of the right lung took place in 9 of our 27 cases of hepatic abscess, or in 33.3 per cent. The invasion of the lung is usually ushered in by an aggravating cough of a convulsive character. The signs of involvement of the right lung are indicated by an increase in the area of dulness at the right base, feeble, tubular breathing and possible increase in the tactile fremitus. In the cases where the pulmonary abscess communicate with a bronchus, the most characteristic feature is the expectoration of a reddish-brown sputum described by Budd as having an "anchovy-sauce" appearance. The peculiar color of this expectoration is very characteristic. The contents of the hepatic abscess itself are not of this character, and the peculiar color is probably due to the admixture of the hepatic pus with blood as it passes through the lung. Microscopic examination of the expectoration will, on persistent search, practically always reveal the presence of amebæ. They were present in the sputum of all our cases. It further will show abundance of disintegrated pus cells, a few liver cells, some elastic tissue from the lung, hematoidin crystals, and occasionally Charcot-Leyden crystals. If the pulmonary abscess at intervals freely evacuates itself through the bronchus, the physical signs over the affected area

may be those characteristic of a pulmonary cavity. The rupture of an hepatic abscess into the lung is usually followed by a decided diminution in the size of the liver as determined by palpation and percussion.

Bacterioscopic and Bacteriologic Examination of Hepatic Abscesses.—In all the cases but one, amebæ were found in the abscess contents. The exception was a case of healed hepatic abscess. Amebæ were found, however, in the pus of a lung abscess, from the effects of which the patient died. The statement that the hepatic abscesses are usually sterile requires to be modified. Not at all infrequently bacteria, in addition to amebæ, are also present. The bacteriologic study of our series of 27 cases gave the following results: The *Staphylococcus aureus* was found in 6; the *Bacillus coli communis* in 5, usually associated with other organisms; the *Streptococcus pyogenes* in 3, and the *Micrococcus lanceolatus* and *Bacillus pyocyaneus* in 1 each.

Avenues of Infection of the Liver.—Two views are held as to how the amebæ reach the liver. One view is that they migrate through the intestinal wall and enter the peritoneal cavity. Once reaching the peritoneum, they are supposed to pass toward the liver, the tissue of which they invade. Councilman and Lafluer considered this the likely mode of infection, and thought it amply explained the occurrence of the large abscesses in the upper part of the liver near the diaphragm, as well as of the multiple smaller superficial abscesses. In support of this view, Lafleur cites a case of dysentery in which a peritonitis existed with amebæ in the exudate over the intestinal coils and over the surface of the liver. Rogers,[11] who has made a study of amebic dysentery and hepatic abscess in India, believes that this is the method of infection in a good many cases.

The most rational theory, and the one that has the largest number of supporters, is that infection of the liver occurs by way of the portal vein. There is

11. Rogers: British Medical Journal, Sept. 20, 1902.

ample evidence in favor of this view. Dr. W. G. MacCallum, of the Johns Hopkins Hospital, in a verbal communication, informs me that in a number of recent cases he has found the amebæ in considerable numbers within the portal capillaries at the margins of the intestinal ulcers. Such a method of infection, it would seem, could alone explain the innumerable small abscesses scattered throughout the entire liver in many of the cases.

2. *Amebic Empyema.*—In two cases the hepatic abscess ruptured directly into the right pleural cavity. In a third case, an amebic abscess of the lung ruptured into the right pleura, giving rise to a pyo-pneumothorax which was recognized clinically.

3. *Perforation of the Colon and Peritonitis.*—Just as in typhoid fever, the intestinal ulceration may become so deep that perforation of the colon eventually results. This occurred in three cases in our series. In one case the patient was operated on within three hours after the perforation occurred. He failed to recover, however.

4. *Intestinal Hemorrhage.*—At first thought it seems somewhat surprising that intestinal hemorrhage is not more frequent, considering the extent of the ulceration in the severest cases. It occurred in 3 cases in the series. Its infrequency is due to the fact that the vessels are thrombosed in the area of infiltration.

Other Associated Infections.—It is of considerable interest that the amebic dysentery was associated with another protozoan infection in a number of instances. Five of the patients also had malaria. Three of these were double tertian and two estivo-autumnal infections. Flexner[12] reported one of these cases and found in addition the *Bacillus coli communis* and the *Micrococcus lanceolatus* in the cultures from various organs. Another case was of extraordinary interest. The patient had suffered from dysentery for six years. On admission it was

12. Flexner: Johns Hopkins Hospital Bulletin, vol. vii, 1896, p. 171.

found that he was also suffering from typhoid fever, confirmed by a positive Widal reaction. The typhoid was complicated by a double thrombosis of the femoral veins. During convalescence he developed an amebic abscess of the liver for which he was operated on, with eventual recovery. One patient had pulmonary tuberculosis.

MORBID ANATOMY.

The intestinal inflammation and ulceration is confined almost exclusively to the large intestine. In 2 of our cases there was slight superficial ulceration in the last few centimeters of the small intestine.

The intestinal lesion consists of ulceration which has been preceded by infiltration of the submucosa due to an edematous condition and to multiplication of the fixed cells of the tissue. In the earliest stage these local infiltrations appear as hemispherical elevations above the general level of the mucosa. The mucous membrane over these soon becomes necrotic and is cast off, exposing the infiltrated submucous tissue as a grayish-yellow gelatinous mass, which at first forms the floor of the ulcer, but is subsequently cast off as a slough. The individual ulcers are round, oval or irregular, with infiltrated undermined edges. The visible aperture is often small compared with the loss of tissue beneath it, the ulcers undermining the mucosa, uniting and forming sinuous tracks bridged over by apparently normal mucous membrane. According to the stage at which the lesions are observed, the floor of the ulcer may be formed by the submucous, the muscular or the serous coat of the intestine. The ulceration may affect the whole or some portion only of the large intestine, particularly the cecum, the hepatic and sigmoid flexures and the rectum. In severe cases the whole of the intestine is much thickened and riddled with ulcers, with only here and there islands of intact mucous membrane. Healing takes place by the gradual formation of fibrous tissue in the floor and at the edges of the ulcers, which

may eventually result in partial and irregular strictures of the bowel.

Microscopic Examination.—A notable absence of the products of purulent inflammation appears. In the infiltrated tissues polynuclear leucocytes are seldom found and never constitute purulent collections. Amebæ are found more or less abundantly in the tissues at the base of and around the ulcers, in the lymphatic spaces, and occasionally in the portal capillaries.

Two kinds of lesions occur in the liver. First there are local necroses in the parenchyma, scattered throughout the organ and possibly due to the action of chemical products of the amebæ. The abscesses, as already stated, are either multiple or single. Multiple abscesses are usually small and generally superficial. In an early stage they are grayish-yellow, with sharply defined contours, and contain a spongy necrotic material, with more or less fluid in its interstices. The larger abscesses have ragged necrotic walls, and contain a more or less viscid, greenish-yellow, or reddish-yellow purulent material mixed with blood and shreds of liver tissue. The older abscesses have fibrous walls of a dense almost cartilaginous toughness. A section of the abscess wall shows an inner necrotic zone, a middle zone in which there is a great proliferation of the connective tissue cells and compression and atrophy of the liver cells, and an outer zone of intense hyperemia. There is the same absence of purulent inflammation as in the intestine, except in those cases in which a secondary infection with pyogenic organisms has taken place. The material from the abscess cavity shows chiefly fatty and granular detritus, few cellular elements, and amebæ in variable numbers, which are also found in the abscess walls, chiefly in the inner necrotic zone. Very frequently the pus in the central part of the abscess contains no amebæ. These are found in gradually increasing numbers as the abscess wall is approached. The organisms remain at the mar-

gins of living tissue, where they can obtain oxygen, which seems essential for their existence.

DIAGNOSIS.

The diagnosis of amebic dysentery depends on the finding of actively motile amebæ in the stools. In the majority of cases the amebæ will be found in the first specimens examined. When the organisms are few in number, and particularly in the chronic cases during the periods of constipation, many examinations extending over days may be necessary before positive findings are made. It is important that the stool should be examined as soon as possible after being passed and while the feces are still warm. In order to prevent the feces from cooling off it is well to have the patient pass the stool into a warmed receptacle and then to immerse the latter in an outer basin containing warm water. Our routine method in the hospital wards, however, has been to secure perfectly fresh specimens of the intestinal contents by the introduction of the rectal tube. When the stools are fluid considerable quantities of fecal matter may be obtained, and when constipation exists small portions of mucus will be found in the lumen of the rectal tube from which preparations for microscopic examination may be made. In searching for amebæ the portions of the feces to be selected are the particles of blood-stained mucus and the small gelatinous masses consisting of necrotic intestinal mucosa. In examining the specimen microscopically, it is well to use the low power first. In this way the slide can be rapidly looked over and the amebæ will be recognized as larger and more refractile than the intestinal epithelial cells and leucocytes from which they have to be differentiated. Even the ameboid movement can be recognized with this power. When a suspicious looking field has been found the high power should then be used. A positive diagnosis of amebic dysentery should be made only when actively motile organisms have been found. When one finds large, round, non-motile bodies containing one or

more red cells one can be reasonably certain that he is dealing with amebæ. A positive diagnosis, however, should be withheld until actively motile organisms are found, which result will almost surely follow in such cases if persistent faithful search be made. There is one type of degenerated epithelial cell which may give rise to confusion. This is an intestinal epithelial cell in which vascuolic degeneration is taking place. The vacuole gradually increases in size at the expense of the remainder of the cell and might be mistaken for the hyaline ectosarc of an ameba. It occupies usually one side and the nucleus is pushed to the other side of the cell. The absence of any ameboid movement should suffice to prevent misinterpretation of these cells. Swollen degenerated leucocytes should give no trouble. The monads are recognized by their peculiar movement and by their different morphology.

When physical signs pointing to hepatic abscess develop in a case of amebic dysentery one naturally suspects that the abscess is of amebic origin. The question of the differential diagnosis between hepatic abscess, subphrenic abscess and pleurisy with effusion will not be considered here. The positive diagnosis of hepatic abscess will depend on the finding of motile amebæ in the contents of the abscess. In a large number of our cases of hepatic abscess contents were obtained for microscopic and bacteriologic examination by means of a long aspirating needle. While some objection to this procedure might be raised, we had no untoward results in any of our cases. The point of introduction of the needle, which should be 8 to 10 centimeters long, will depend upon the suspected position of the abscess. The procedure may be repeated a number of times if the first puncture is negative. The absence of amebæ in the contents does not prove that the abscess is not of amebic origin, for it is a frequent occurrence in cases operated on not to find amebæ until the abscess evacuates itself and until particles of the abscess wall appear on the

dressings. The absence or partial absence of pus cells in the contents of an hepatic abscess is always suggestive of its being amebic in origin, because the ameba is not a great pus producer. The bulk of the contents consists largely of granular debris resulting from broken down liver cells. An amebic origin should always be suspected when the cultures prove negative. Amebic abscesses of the liver are not by any means always sterile, however, as has been already amply shown in this series.

When a patient with an hepatic abscess suddenly begins to expectorate quantities of sputum possessing a reddish-brown "anchovy-sauce" appearance, the establishment of an hepato-pulmonary abscess must be considered. The granular character of the pus on microscopic examination and the finding of liver cells and hematoidin crystals confirms this suspicion. Careful and persistent examinations of the sputum will nearly always reveal the presence of amebæ when the hepato-pulmonary abscess is of amebic origin. Amebæ were found in the sputum in all our nine cases in which this complication occurred.

After the discovery of *Bacillus dysenteriæ* some investigators were at first inclined to believe that this organism was the primary agent in setting up the intestinal inflammation and that the *Amœba dysenteriæ* was a secondary invader and of minor pathologic importance. These observers now credit the ameba with full powers to produce the lesions which are characteristic of amebic dysentery. The blood serum of nearly all the cases of amebic dysentery admitted to the Johns Hopkins Hospital during the last two years has been tested in order to ascertain whether it possessed any agglutinative action with the *Bacillus dysenteriæ* obtained from various sources. The test was invariably negative, thus tending to show that *Bacillus dysenteriæ* played no part in the production of the disease in our cases.

COURSE AND PROGNOSIS.

Any one seeing numerous cases of amebic dysentery learns to be guarded in his statements to the patient concerning the ultimate outcome of the disease. Tendency to a relapse of the dysenteric symptoms is one of the striking characteristics of the affection. In the majority of cases the dysentery yields fairly readily to absolute rest and intestinal medication in a very few days, and the bowels may become constipated and move only when the medicated irrigations are administered. The recurrence of the symptoms in patients discharged from the hospital apparently cured, is no doubt due to the failure of the intestinal irrigations to reach the living amebæ buried deep in the cells of the submucosa of the intestines. These organisms that survive eventually multiply in sufficient numbers to again set up the local intestinal ulceration with a consequent relapse of the symptoms.

That the disease is a grave one is amply illustrated by the mortality in the Johns Hopkins Hospital series. Of the 119 cases, 28 or 23.5 per cent. terminated fatally. Hepatic abscess is a very serious complication. Of the 27 cases with this complication 19 died. Five recovered after being operated on. Two of the 9 cases of hepato-pulmonary abscess left the hospital very much improved with good prospects of recovery, although their ultimate fate is not known. One case of abscess refused to remain in the hospital and was not treated. The seriousness of the hepatic abscess as a complication is further emphasized by the fact that the 5 recoveries after operation were out of a total of 17 cases operated on.

TREATMENT.

Until we know something definite of the life history of the ameba outside the human body it will be difficult to prescribe any adequate prophylactic line of treatment. Presumably, however, the same precautionary measures against typhoid infection, especially in

connection with drinking water, will prove serviceable in this disease.

When the disease has actually established itself, the patient should be put to bed and kept absolutely quiet until the active symptoms have been controlled. The diet should be simple and easily digestible. A long list of medicines, administered by mouth, has been tried without satisfactory results. Even ipecac, formerly so strongly recommended in the tropics for the various forms of dysentery, has fallen largely into disuse. Local treatment gives the best results. Various astringent solutions have been advocated and may be tried. Large high irrigations of a solution of quinin have given the best results and this has been the routine treatment in our wards. The success of the treatment depends largely on the method of giving the irrigation. It must be remembered that the rectum in the majority of cases is not at all or only slightly involved and that the ulceration is most extensive in the cecal pouch and in the ascending colon. One must insure, then, the irrigation fluid reaching the upper part of the colon. This is best attained by having the patient's hips well elevated on firm pillows and to have him roll occasionally from his left to his right side. The same object may be secured by placing the patient in the knee-chest position. A liter of the quinin solution should be allowed to run into the colon twice daily. The strength of the solution at first should be 1-5000, gradually increasing it until it reaches 1-1000 or 1-500. In some cases we have given the patients at intervals an irrigation of a weak solution of hydrogen peroxid with apparent benefit. Colostomy has been recommended very highly by some in the intractable cases. It was only performed once in our series. The patient was almost moribund at the time of the operation and died shortly after.

Where the dysentery is complicated by hepatic abscess, the latter should be operated on at the earliest possible opportunity after a diagnosis has been estab-

lished. Five of our 17 cases operated on recovered. While these results are fairly satisfactory they leave much to be desired. The earlier the operation the better the patient's prospects will be. The unsuccessful results after operation have, in many cases, been shown to be due to the fact that the abscesses are multiple and consequently only one abscess is evacuated, the others continuing to exert their injurious effects after operation. The operation is usually done in two stages in this hospital. The first operation is to promote shutting off the peritoneal or pleural cavities, depending on the seat of the incision. The second operation is for the evacuation of the abscess, and is performed a few days later. This delay, of course, is only permissible when the patient's condition will allow it. Rogers advocates very strongly a quinin solution of about 1 per cent. strength for irrigating out the abscess from time to time. He believes that the amebæ in the abscess wall are more quickly destroyed, thus allowing of a more rapid healing.

Where the abscess opens into the pleural cavity with the establishment of an empyema, free drainage must be established. When intestinal perforation occurs the experience with typhoid perforation in recent years leads one to hope that success might follow operation if performed very early.

SUMMARY.

In nearly fourteen years 120 cases of amebic infection have been admitted to the Johns Hopkins Hospital. Of these, 119 were cases of amebic dysentery or amebic abscess of the liver, the remaining case being one of amebic abscess of the floor of the mouth. Of the 120 cases 118 were admitted to the medical wards. During this time there were 15,817 medical admissions, the amebic dysentery cases constituting 0.74 per cent. of the cases. Of the 119 cases of dysentery, 82 were apparently contracted in Baltimore and 13 in the State of Maryland outside Baltimore, or a total of 95 cases

within the borders of Maryland. The largest number of cases occurred in the third decade. The youngest patient was a child two years and eight months old, and the oldest a man of 71. A point of special interest is the comparatively large number of cases in children under 10 years. Eleven cases, or 9.4 per cent., occurred in the first decade, 6 of these belonging in the first hemi-decade. There were 108 males and 11 females. The series comprised 107 whites and 12 blacks, or a proportion of 9 to 1. The total ratio of whites to blacks in the hospital is about 7 to 1. A secondary anemia of moderate grade occurs. Occasionally there is a polycythemia. The average leucocyte count in the uncomplicated dysentery cases was 10,600. In the hepatic abscess cases it was 18,350. Hepatic abscess occurred in 27, or 22.6 per cent., of the cases. Nine of these ruptured into the right lung and 2 into the right pleura. Three opened into the inferior vena cava. In 10 cases there was a single large abscess in the upper part of the right lobe. In 8 cases there were multiple abscesses. Perforation of the colon with peritonitis occurred in 3 cases; and severe intestinal hemorrhage in 3 cases. Five cases were complicated by malaria; 1 by typhoid; 1 by pulmonary tuberculosis; and 1 by a strongyloides intestinalis infection. The agglutinative blood reaction with *Bacillus dysenteriæ* was negative in all cases tried. A marked feature of the disease is the great tendency to relapse. The mortality was high; 28, or 23.5 per cent., terminated fatally. Hepatic abscess is a serious complication, 19 of the 27 cases terminating fatally. Five were cured out of 17 operated on. Two of the hepato-pulmonary abscess cases were discharged very much improved. One abscess case did not remain to be treated. Quinin irrigations still seem to give the best results in the treatment of the dysentery.

THE TREATMENT OF ACUTE DYSENTERY.

J. M. ANDERS, M.D.
Professor of Medicine and Clinical Medicine, Medico-Chirurgical College.
PHILADELPHIA.

The subject will be considered under three heads: 1. Regiminal Treatment; 2, Internal Remedies; 3, Topical Applications.

I. REGIMINAL TREATMENT.

The regiminal treatment, properly carried out in its various details, is potent to render the course of this affection distinctly milder and more favorable. Among the eminently important measures in bacillary dysentery I should regard rest in bed throughout the attack as deserving of a prominent place, and it should be rigidly enforced in all instances, not excepting the milder sporadic cases. As a means to conserve the bodily strength and energy, absolute repose is never uncertain in its effects; its extreme importance to the inflamed and ulcerated bowel, with a view to promoting resolution and the healing process, is universally acknowledged. The efficiency of rest in bed can be appreciably enhanced by constant, careful attention to the ventilation of the sick chamber. Fresh, pure air, without strong currents, is practically important as an aid in preventing the development of dangerous asthenia.

Too close attention to the diet and manner of feeding can not be paid. Certain dietetic principles need to be kept in view. For example, it must be recollected that concentrated, soft and liquid forms of food tend to

favor constipation, whereas bulky, hard articles stimulate intestinal peristalsis.

Our aim in this disease is to minimize the intestinal contents, and thus, also, the amount of decomposable material that will reach the seat of the morbid changes. To reduce the putrefactive processes in the intestines, especially during the active period of acute bacillary dysentery, an absolute liquid diet composed principally of milk and albumin water, is a prime requisite. The milk should be predigested and given in small quantities, and the dejecta carefully scrutinized; and the appearance of food remnants in the feces is an indication either for further dietetic restriction or a change of pabulum. Dieting must be systematically conducted. When not objected to, the albumin water is admirably suited to this disease in the early acute stage. If carefully carried out, a milk diet, however, is attended with the least degree of intestinal putrefaction. Nothing is more pernicious in its effects upon the local lesions than to overburden the much-enfeebled digestive organs.

It is doubtless true that the fats increase intestinal putrefaction, and must be allowed in relatively small amounts in this disease, if at all. This is probably also true of carbohydrates, although Bachman's experiments, contrary to the general teaching, show that they exert no marked influence on intestinal decomposition. Nutrients are of signal importance in supporting the vital powers, and may be preventive of dangerous asthenia. Finally, not all foods, even among the blandest and most nutritious forms, are equally well borne by the stomach, and several changes may be necessary during the course of a given case.

I have dwelt somewhat at length on the subject of diet in dysentery, believing that there is a lack of appreciation on the part of the general profession of its true value in the management of this disease.

Stimulants are required in the severer forms of endemic, and more especially epidemic, dysentery, to over-

come the pronounced asthenic which often develops at a comparatively early period of the disease. Marked and even alarming circulatory enfeeblement may be met, and this calls for the free use of diffusible stimulants (e. g., champagne). In extreme adynamia, strychnin should be employed subcutaneously; and the timely, judicious employment of the normal saline solution is highly serviceable in similar cases. It has seemed to me that in our eagerness to find a special method of treatment (of which there are not a few from which to make a selection), agencies and elements whose efficiency has been satisfactorily demonstrated by the experience of by-gone generations of physicians, have been omitted from consideration in discussing the management of dysentery. An examination of the literature will confirm this opinion, so far as relates to the use of stimulants in this disease at least. The regimen, during the convalescing period, should receive much attention. Indiscretion in diet must be obviated; the too early use of fats, fruits and the coarser vegetables often cause a return of the dysenteric symptoms. The habits of the patient must be vigilantly looked after until full health is restored, and an abdominal bandage of wool is advised following an attack of dysentery, and may be warmly recommended.

The prophylactic measures applicable in the treatment of dysentery are much the same as those demanded by other micro-organismal diseases, notably typhoid fever, and need not be detailed here. Especial attention, however, should be paid to the disinfection of the excreta, while the drinking water must be both filtered and boiled. Unless a rigid system of disinfection of the stools be carried out during the prevalence of an epidemic, an infected individual contributes to the common source of contamination.

2. INTERNAL REMEDIES.

It is customary to speak of special methods of treatment by medicinal agents. This is unfortunate, since it

tends to lead to neglect of other most important elements of the treatment. Pre-eminent among special drugs stands ipecacuanha, and although a most valuable agent in this disease, it can not be said to possess an abortive, or even curative, action; it is not a specific for dysentery. Says Hare:[1] "So far as we know, any influence which it possesses is brought about by reason of the fact that it tends to produce a rather profuse flow of bile in such patients, and the close connection which exists between the torpidity of the liver (to use a popular phrase) and congestion of this organ, with congestion of the colon, makes its employment under these circumstances more rational than would appear at first glance." Woodhull,[2] however, claims that "ipecac is not, as commonly supposed, a depressant or an evacuant, except in a secondary sense. Its primary action is that of a stimulant more directly of the organic nerve." He believes that this drug thus contracts the intestinal capillaries. Treatment, therefore, should be prompt.

R. Kobert[3] states that the antidysenteric action is supposed to reside in a glucocid, ipecacuanhic acid. It is probable that the use of ipecac has been less enthusiastically supported by clinicians in recent times than was formerly the case, because its mode of administration has not been of such character as to insure the highest efficacy of the drug. Kieffer[4] gives the following method, which has been adopted in tropical dysentery: "Nourishment of every description is withheld for three to four hours. Then 15 to 25 drops of tincture of opium are administered and a small sinapism applied to the epigastrium. In from twenty to thirty minutes, when the effect of the opium begins, from 30 to 60 grains of ipecac are administered in powdered form stirred up in one or two ounces of water. The patient lies on his back in bed with both eyes covered with damp, cool com-

1. Proceedings of the Phila. Co. Med. Society, Sept., 1902, p. 51.
2. Therapeutic Gazette, April 15, 1902.
3. Ther. Monatsch., August, 1902.
4. Tropical Dysentery, Philadelphia Med. Jour., Jan. 31, 1903.

presses. The pillow is removed. The patient is carefully enjoined to make no movement. If mucus gathers in the mouth and throat, a nurse, on sign from patient, removes it with a cloth or towel. If all these precautions are rigorously carried out, the patient retains the dose."

It requires at least two hours for ipecac to produce its effect. If it be vomited within an hour, the dose is repeated as soon as the nausea subsides. When all signs of nausea have disappeared the feedings are to be resumed. The next day repeat the dose of ipecac, diminishing it by 5 or 10 grains, and continue in this way for three or four days, using always the same precautions against vomiting. Kieffer feels that if administered in this way, ipecac will prove of value in the dysentery of the United States. Although aware of the fact that most authors recommend the massive dose, in my hands quarter-grain doses administered every half hour have in some cases seemed to be quite as effective, and in children the smaller doses are to be preferred. Strassberger[5] lauds ipecac highly in the treatment of the acute exacerbations of amebic dysentery, using one dram of the Rio article, with a full dose of opium, twice daily. The effect was prompt and permanent, and in only one instance did a real relapse occur. Upon further investigation and observation he found that while it may be a specific in amebic dysentery, it has little or no effect on the bacillary forms.

The use of saline laxatives in the treatment of dysentery is generally adopted. The best preparation is magnesium sulphate, which produces an eliminative effect, depletes the intestinal mucosa, and at the same time inhibits undue peristalsis. It is best administered in doses of one dram, diluted, every hour until free purgation is induced. This is followed by the generous exhibition of acid. aromat. sulphuric., which is given partly for its astringent effect and partly to render the intestin-

5. Münch. Med. Woch., Sept. 9, 1902.

al contents acid. It has been shown by Cunningham of India, with reference to amebic dysentery, that if the contents of the intestines are alkaline, the amebæ thrive and multiply, while conversely, if the intestinal matter is rendered acid, their multiplication ceases for want of a suitable medium. The combined saline and acid method of treating dysentery rests on a most rational basis. I have, however, found and elsewhere expressed the opinion that if saline purgatives are employed at a late stage of the affection, or even after the full development of a given case, they are invariably attended with baneful effects. Richmond[6] also observes: "It is of the utmost importance that cases in which the sulphate of magnesium or sulphate of sodium should come under treatment early in the attack, before much ulceration has taken place, otherwise the septic absorption continues. In some cases treated by this method at Duelfontain, little or no improvement accrued, and the "patient died in a condition of toxemia, with rapid failing pulse, delirium and profuse diarrhea, with much painful tenesmus." Opium in some form, preferably as Dover powder, is promptly effectual in meeting certain leading systematic indications, as pain irritability, restlessness and undue peristalsis. For intensely distressing tenderness, either an opium suppository or laudanum by enema exercise a beneficial effect. The preparations of bismuth are valuable, particularly in the milder sporadic cases, and in the advanced stage of the severest forms; they may be advantageously combined with Dover powder and an antiseptic substance.

Phelm,[7] who has had an extensive experience in the treatment of this disease in Africa, advises the successive use of purgatives, calomel and subnitrate of bismuth. A suspicion of dysentery calls for 30 grams of castor oil. If the case be one of dysentery, calomel is given after twelve to eighteen hours. At the end of

6. London Lancet, June 15, 1901.
7. Deutsche Medicinische Wochenschrift.

three days, calomel is succeeded by bismuth subnitrate, which, in turn, is kept up for three or four days, or until the stools become solid again. It may be questioned whether any of the intestinal antiseptics exercise a potent influence; and it may be safely assumed that they are without the power to destroy the infecting micro-organism. The antiseptic care of the intestinal tract in bacillary dysentery, however, should be one of the aims of treatment, and guaiacol, salol and the preparations of naphthol are the agents of choice for this purpose. They may be combined either with the ipecac or salines, or with both. Richmond,[8] during an experience of the treatment of dysentery, found that none of the recognized methods were infallible, and the use of sulphur in the treatment of this complaint was suggested to him on the analogy of the treatment of anthrax by sulphur, as advocated by Mr. Arbuthnot Lane.[9] He had under his care in the Imperial Yeomanry Hospital at Pretoria, 20 acute cases and 10 cases of chronic diarrhea following attacks of acute dysentery. All the acute cases were treated with sulphur and Dover powder, and the chronic cases with sulphur alone, and in every instance a cure resulted. The ameba was carefully searched for in all of Richmond's cases, but was never found, and he believes it to be a justifiable speculation that the dysentery of South Africa was, like that of Japan and the Philippines, due to the Shiga bacillus. This observer prescribed 20 grains of sublimed sulphur combined with 5 grains of Dover powder four-hourly in the acute cases, and a like dose of sulphur alone in the chronic diarrhea resulting from acute attacks. The signal value of this remedy has been confirmed by the experience of other competent clinicians, both abroad and in this country. Although the exact mode of action of the sulphur is unknown, it is probable that the resulting sulphuretted hydrogen and sulphur acids inhibit the growth and pro-

8. London Lancet.
9. The Medical Week, Dec. 8, 1891.

pagation of the *Bacillus dysenteriæ*. Stengel has observed the same curative effect in amebic dysentery, and it is quite reasonable to suppose that the modus operandi of its action is similar in this form to that of bacillary dysentery. This remedy is strongly advised on theoretical grounds, and in view of the brilliant results already reported, is worthy of a more extended trial than has as yet been accorded it. It will probably be found to be best suited to protracted or chronic forms of the complaint. Dabney has pointed out the eminently important fact that the existence of certain complicating conditions, as malaria and various intestinal worms, may occasion non-success in attempts at treatment, unless recognized and met by the use of quinin or anthelmintics according to the given case. Failure of the ipecac treatment may also be due to the unreliability of its preparations.

Another method of treatment is by the use of antidysenteric serum. Gay[10] has prepared vaccines of uniform activity, and by means of which he could protect guinea-pigs from a multiple fatal dose of living dysentery bacilli, and also produce in the horse an active, immune serum. Gay thinks that a useful serum therapy of bacillary dysentery and of certain forms of the summer diarrheas in infants (the latter having recently been shown to be due to the *B. dysenteriæ*) is rendered highly promising. Shiga has successfully employed a serum which he himself has prepared. It is both protective and curative, but not as yet available for general use.

3. TREATMENT BY TOPICAL APPLICATIONS.

The treatment of dysentery by high intestinal irrigations is doubtless of value, but quite a few recent writers are not ardent advocates of the use of large enemata in acute bacillary dysentery, believing that they may cause atony and weakening of the coats. Astringent and antiseptic solutions are used for this purpose; they must be weak, otherwise the tormina and tenesmus—distress-

10. Univ. of Penna. Medical Bulletin, Nov., 1902.

ing features of the disease—will be materially increased. The method is better adopted to the more chronic forms of dysentery, the amebic variety in particular. In the latter, a warm solution of quinin (1-2,000, 50-5,000) is almost universally acknowledged to be more or less efficacious. It may be questioned, however, whether the amebic form is curable by known methods of treatment. In the milder forms of acute bacillary dysentery, copious enemata are unnecessary. If employed in this class of cases, the same precautionary measures must be taken as in the severe types of the disease.

Among the remedies thus employed are silver nitrate, strength of solution (gr. ss. to 1 ad ℥i); salicylic acid (1 to 2 per cent.); tannic acid (½ to 1 per cent.); zinc. sulpho-carbolate (gr. i-ii ad ℥i); and mercuric chlorid (1-6,000).

It has been my custom to alternate an astringent and antiseptic solution, administering a copious clyster at twelve-hour intervals.

The mode of giving the irrigations is of vital importance. A fountain syringe should be employed, and as shown by the experiments of Hare and Martin; the pressure should never exceed two or three feet. Intestinal lavage must be a slow process in order to be successful, and to this end the flow should be interrupted at short intervals. The patient during the enemata should assume either the dorsal position or that on the left side, with the hips elevated; or if he be not too ill, he may be turned from side to side during its administration.

In conclusion, I would emphasize the fact that it is impossible to indicate a special mode of treatment that would be suitable for all cases of dysentery since each case, whether of the bacillary or amebic variety, presents certain individual peculiarities. The therapeutic measures must vary also with the particular stage of the affection and the previous condition of health of the patient.

DISCUSSION

ON PAPERS OF DRS. MASON, KNOX, BURNS, HARRIS, FUTCHER AND ANDERS.

DR. ALEXANDER LAMBERT, New York—This is not a common disease in the North. Still, it is not uncommon among transients going through New York and who land in Bellevue Hospital. Some years ago I was surprised to find cases of amebic dysentery developing in people not 25 miles from New York; I have had four such cases in the past five or six years. It has been stated that liver abscesses were more frequent in the North than in the South. So far as a limited experience in a number of cases of amebic dysentery is concerned I have seen abscess develop in a large number.

I have found that high enemata given one week, and quinin the next week, and silver once or twice a day, have given me better results and quicker improvement than followed when I persisted in quinin or silver. In the more chronic cases I have given 1-500 bichlorid solution and run it down to 1-1000 with beneficial results. Salol and castor oil seem to give more satisfaction with some patients. Most of these cases have gone out of the hospital cured unless they died of liver abscess.

In all these cases of amebic dysentery there was one symptom that I have been unable to find mentioned in any textbook, viz., the severe amount of pain in the muscles at night; painful cramps in the muscles were found in every case of amebic dysentery. In the other cases of dysentery this was not complained of. I should like to hear from the Section on this symptom.

DR. T. S. DABNEY, New Orleans—At first I used sulphate of magnesia, but experience has taught me that sulphate of soda by reason of its action on the liver is far more efficacious. I also wish to state that the combination I use has a little morphin added, 1/6 to 1/32 of a grain to the dram; there is also dilute hydrochloric acid and peppermint water, and this combination acts well. If you take six cases of dysentery and use sulphate of soda in three, and sulphate of magnesia in the other three, you will not use sulphate of magnesia again. In conjunction I use acid drinks of dilute sulphuric acid, or nitrohydrochloric acid. All my patients drink this all the time. Experience has taught that the bacillus of dysentery seems to thrive poorly in acid and acid medium; it seems to require an alkaline medium, hence acidulated drinks inhibit the growth of this bacillus.

While in Colombia, South America, in the summer of 1892, I was asked by Dr. Angoula, a native Colombian physician, a close observer of the endemic diseases of his country, to visit the village of Rio Frio, situated in the midst of vast banana haciendas, where a disease, characterized by severe in-

testinal hemorrhage, was at that time prevailing. In almost every thatched hut we found patients suffering from profuse bloody discharges at frequent intervals. A moderate amount of fever, but not much tormina or tenesmus was present. So great was the loss of blood that the patients seemed exsanguinated. On being asked by the Doctor for the diagnosis and treatment, I politely declined either, and remarked I was the student and he the teacher. His diagnosis was intestinal paludic fever and his routine treatment one gram of the sulphate of quinin dissolved in water acidulated with dilute sulphuric acid, this to be repeated every four hours until the character of the stools markedly changed. On our second visit, six hours later, we found the diagnosis verified by the treatment, for never in my professional life before had I seen such immediate and happy results follow so promptly on the administration of any remedy. I am inclined to believe the acid medium played an important part in the cure.

Dr. T. J. Moore, Galveston, Texas—I believe that there is not only amebic but also bacillary dysentery where I live. During the past four years we have carefully examined every malarial fever case, and, as a result, I am not ready to admit that we have malarial dysentery. I have treated during the past four years several hundred cases of malarial fever and associated with many there has been dysentery, but on closer investigation we have found, in probably all of them, some other cause for the dysentery than the malarial infection. I would rather regard dysentery as one of the symptoms of malarial fever seen occasionally. Hemoglobinuric types of fever with dysentery seem to be more common. We must admit that there is some gastrointestinal disturbances in many cases, but I am not ready at all to admit that we have malarial dysentery. Many cases of amebic dysentery get better on quinin and some better without quinin. During the past three years, at my suggestion, in the wards of the hospital we have been using the fluid extract of chaparro, a Mexican shrub. In the series of cases in which this drug was used, some ten or twenty, there was a rapid disappearance of the dysentery, and also of the amebæ from the stools. This drug is well worthy of trial.

Dr. Lambert has referred to cramps in the muscles of the arms and legs; this is especially true in the severe and acute cases where you find a great number of these amebæ, many containing red blood cells. In the severe cases the amebæ show a disposition to collect within themselves a large number of red blood cells. In the acute form there is another feature, the acute swelling of the mucous membrane which may cause a complete occlusion of the intestines.

Dr. Walter Sheopshire, Yoakum, Texas—The cause of this disease has been to me an enigma for many years. Three

years ago I had a case in a child nine months old. Severe symptoms were referable to the lower rectum and the disease progressed rapidly. An examination of the feces disclosed amebæ which contained red blood cells. Irrigation of the rectum and colon with quinin caused a disappearance of the ameba. When first examined I found the colon bacillus in large quantities. I called another physician in and he agreed with me in my diagnosis. The colon bacilli persisted, and the disease continued until the child began to vomit. In the vomit were found numbers of these bacilli, but no amebæ. At the autopsy, macroscopically, there was noted the characteristic undermining ulcers of the amebic dysentery. Further up in the tract were found bright red congestion of fresh inflammation. Microscopically there were found the same bacilli throughout the intestinal tract, in the mesenteric glands, liver and spleen. We then decided that this bacillus was the cause of the disease. Specimens were sent to Dr. Smith of Galveston, and he agreed that they were the colon bacilli, but said they were harmless in the intestinal tract. I am firmly convinced, though, that they were the cause of the disease. Since then I have not been able to discriminate between the two, Shiga's and the colon bacillus, and shall appreciate an accurate method of doing so.

Dr. Charles F. Mason, Ft. Sam Houston, Texas—A great many soldiers come to me after they have passed through several hospitals and after they have been recommended for discharge on account of disability; now, after a little experience with the following treatment, we find that these men can be much benefited. First, an anesthetic is given and the sphincter thoroughly dilated in order that a careful examination of the mucous membrane of the rectum can be made; almost always we find a little ulceration in the rectum within five or six inches from the sphincter. These ulcers are apt to be undermined, forming little pouches in the mucous membrane in which a probe can be introduced or hooked, and in which foreign matter as it comes down the bowel may gain entrance. Such ulcerations are treated by slitting them down to the bottom and curetting them or touching them with pure carbolic acid, or both. The bowel is then washed with a normal saline solution and the patient placed in bed and kept quiet on a liquid diet and preparations of milk. Irrigation with normal saline solution are used thereafter once or twice a day. Every case with one exception has been cured and returned to duty.

Dr. J. H. M. Knox, Baltimore—The work was begun last summer at the Thomas Wilson Sanitarium with much skepticism as to the relationship of the Shiga bacillus to the diarrheal cases under treatment. The dysentery organism was isolated in case after case which seemed to be quite identical

clinically with the several types of intestinal disorder in infancy which are grouped together as summer diarrhea. The claim is not made that every case of diarrhea in infancy is produced by this bacillus, but our experience at Baltimore last summer would certainly indicate that a large number at least of both the acute and chronic diarrheal disorders of infants are associated with *Bacillus dysenteriæ* (Shiga). It is to be emphasized that the isolation of this organism from the stools or intestinal tract requires some special skill and a bacteriologist well trained in other lines may easily overlook the specific bacillus, as indeed must have been repeatedly done.

It is to be hoped that during the coming summer a very large series of cases will be carefully studied in many localities that it may be determined to what extent the Shiga bacillus is responsible for the widespread infant epidemics which occur each summer. I should like to urge that all those who see these cases and have access to bacteriologic laboratories familiarize themselves with the special technic necessary in isolating the Shiga bacillus and then join in the effort to assist in the bacteriologic study of summer diarrhea. We shall be glad to send cultures from the sanitarium to any who wish to familiarize themselves with the organism.

Dr. Wm. Britt Burns, Memphis, Tenn.—I do not think we are any nearer a definite knowledge of the etiology of dysentery. I am aware that most all authorities at the present time do not agree that malaria has been a causative factor in dysentery. I am also aware that some cases of bacillary and amebic dysentery occur when in unison with the malarial parasite in the blood, but I do not find that those cases respond to treatment rapidly, and I do not believe that the ordinary bacillary and amebic dysentery would respond so readily to the treatment, as I have seen many cases do under the treatment ordinarily used in malarial disease.

Dr. H. F. Harris, Atlanta, Ga.—I wish to call attention to the treatment of amebic dysentery with hydrogen dioxid, concerning which, I observe, nothing so far has been said. When, in 1891, I saw my first case of this disease, I used the treatment first recommended by Loesch, and since lauded by various writers, which consists in injecting quinin solutions into the large intestine of those afflicted with the malady in question. Although the patient showed absolutely no improvement, I employed the same treatment in the next nine cases that came under my observation, and not in a single instance was there any appreciable effect produced. This led me to make some investigations as to the power of sulphate of quinin to kill the *Amœba coli*, and I was much astonished to find that this substance seemed to exert but little influence on these parasites. In one instance the amebæ remained alive at the temperature of the room for more than

twenty-four hours in a supersaturated solution of this substance. It was thus clear that quinin sulphate could not be relied on to produce the death of the amebæ, particularly when they are embedded in the tissues, nor can this substance destroy the numerous bacteria that lie in and around the ulcers and are doubtless largely responsible for many phases of the clinical picture in this disease. Failing to get any results from the quinin injections, I tried almost everything that has been recommended in the treatment of chronic dysentery, and never succeeded in securing results in any way satisfactory. Finally, at the suggestion of a student, I used daily injections of hydrogen dioxid, diluted with water from four to six times, with the result that there was an immediate and most striking amelioration of the symptoms in almost every instance—in every case, I believe, where the solution was brought in contact with the lesion. A remarkable result of this treatment is that blood is never seen in the stools after the first injection in instances where the medicament acts properly. The treatment is continued daily for a week, and after this the number of injections are gradually reduced in number until at the end of from four to six weeks no more are given. Especially at first the injection sometimes causes severe pain, and when this is the case it is well to administer morphin an hour before the treatment is given. It is absolutely necessary that the patient be placed on a strict milk and egg albumin diet, and that he should remain in bed for from two to four months. Occasionally patients recover when these precautions are not taken, but it is unusual. I have only seen muscular cramps in bad cases.

Dr. T. B. Futcher, Baltimore—I do not recall that any of our patients complained of muscular cramps. This symptom may occur in the southern states, where a severer type of the disease probably prevails.

We have to modify our views considerably concerning the bacteriology of the amebic hepatic abscesses. Cultures have shown that the abscesses are sterile in a much smaller percentage of cases than was formerly believed to be the case. The more frequent organisms are the *Staphylococcus pyogenes aureus* and the *Bacillus coli communis*. The *Streptococcus pyogenes* was present in three of our cases and the *Micrococcus lanceolatus* and *Bacillus pyocyaneus* once each.

In the line of treatment quinin irrigations yielded the best results. The success of this treatment depends largely on the way the irrigations are given. The object sought for is the assurance that the quinin solution reaches the cecum and ascending colon, where the ulcerations are most extensive. The relapses are due to the failure of the drug to reach the amebæ buried in the deeper layers of the submucosa. In one case an

infusion of a Mexican herb which had been sent us was tried with fairly satisfactory results.

Dr. James M. Anders, Philadelphia—It is probably advisable to reiterate the importance of the benefit to be derived from absolute rest until all of the acute symptoms have subsided, whether for a shorter or longer period. The question of diet in this disease is one to which too little attention has been paid. I believe that ipecacuanha is the best single remedy for treatment of this disease, but it is not a specific, and not all patients can take massive doses. One-fourth to one-half grain doses at short intervals will give excellent results in many cases. Saline treatment should not be employed in advanced cases, but only when the patients come under observation early; as the result of a considerable experience with this method I find that if employed in the advanced stages of dysentery it aggravates rather than benefits the condition. The sulphur treatment should especially be given a trial in those cases in which ipecacuanha fails. This agent seems to be well adapted for the subacute or chronic forms; better than to the acute, bacillary form of the disease.

ABDOMINAL PAIN IN PLEURISY AND PNEUMONIA.

JAMES B. HERRICK, M.D.
CHICAGO.

That a disease in one part of the body may call forth painful sensation in another part is a matter of everyday observation. Pain in the knee may mean hip-joint disease; the passage of a renal calculus may make pain in the testicle or penis; a gallstone may cause pain referred to the neighborhood of the right scapula; pain in the neck or left arm and hand may indicate angina pectoris and coronary sclerosis. While in pleurisy and pneumonia the pain is usually referred to the chest and to the side affected, it may be referred to points remote from the site of the disease, as to the opposite side or to the abdomen. This fact had not escaped the keen observation of our forefathers in clinical medicine. Laennec[1] speaks of the pain of pleurisy as sometimes shifting its place and "occasionally," he says, "from the beginning of the disease, we have a stitch on the right side and pleurisy on the left." Gerhardt also reports this fact. Huss[2] treated of this subject, explaining the pain on the other side by the occasional anastomosis of right and left intercostal nerves. Barnard,[3] who has lately written of abdominal pain in pleuropneumonic diseases, cites Andral, Watson and Fagge as clearly recognizing

1. Laennec: Treatise on Diseases of the Chest, etc., Forbes Trans., p. 450.
2. Huss: Deut. Archiv f. kl. Med., ix.
3. Barnard: The Simulation of Acute Peritonitis by Pleuropneumonic Diseases, London Lancet, clxiii, Aug. 2, 1902, p. 280.

the fact that severe abdominal pain might have its origin in the pleura, and be misleading unless care were exercised in the physical examination. While several articles have lately called attention to the danger of confusing pulmonary and pleural diseases with peritoneal, this danger is not touched upon by many writers on diseases of the chest or abdomen, nor has it among practitioners of medicine and surgery the general recognition that it deserves. But the subject is well worth emphasizing. For in these days, when even the recent graduate stands ready to attack the appendix of every individual the moment there is right iliac pain, tenderness and rigidity, with fever, the seriousness of a mistake in diagnosis in a case of this sort is plain. This, then, is my excuse for presenting a subject that is not new.

The lower six intercostal nerves—the anterior divisions of the dorsal nerves—supply the abdominal wall, as well as a part of the parietal and diaphragmatic pleura. An irritation, e. g., from inflammation or pressure, in the course of one of these nerves might readily cause a pain that would be referred to the distribution of this nerve, i. e., to the abdominal wall. In cases of pleurisy how much of the irritation is produced by true neuritis is, perhaps, uncertain. By many intercostal neuritis is regarded as of common occurrence in pneumonia and pleurisy.[4] The eleventh nerve is distributed over the iliac region. Pain here would easily make one think of the appendix if on the right side, or if on the left, perhaps of the rarer left-sided appendicular pains. The other nerves would cause pain referred to the umbilical, epigastric or hypochondriac regions and the confusion that might result when we think of the possible significance of such pain in the way of ulcer of the stomach, gall bladder mischief, pancreatitis, etc., is clearly seen.

---

4. Fraentzel: Ziemssen's Encyclopedia, Amer. Trans., iv, p. 632, inclines to the view that was held by Beau that intercostal neuritis is a common accompaniment of pleurisy.

This close anatomic nerve connection between the abdominal wall, the pleura and the intercostal muscles is indicative of a physiologically intimate relationship, clearly seen in the common function of the thoracic and abdominal muscles in the act of respiration. Hilton's well-known law declares that the interior of a joint, the muscles moving it and the skin over the muscular insertions are supplied by the same nerve trunks. This law also applies to the pleura, which, he says, can be compared to a joint. And the muscles to be regarded as moving the joint are not alone the intercostals, but the abdominal as well.[5] Not only may pain of pleural origin be referred to the abdomen, but in their effort to lessen pain and to give the joint, i. e., the pleura, rest, the muscles remain quiet and may even be tense. One can readily understand how the tenseness of abdominal muscles, added to pain, may be deceptive because of the close resemblance to the phenomena of abdominal disease. And how strikingly like the picture of abdominal inflammation may be the combination of pain and rigidity when chilliness, fever, rapid pulse, etc., are also present.

There is still another possible explanation of some of the abdominal disturbances that are seen in pleurisy and pneumonia. The phrenic nerve in its course through the chest lays itself open to the attack of a pleurisy involving the pericardial, diaphragmatic or costal surfaces. This nerve is not solely motor. Henle, Luschka and others regard it as a mixed nerve. Van Gehuchten[6] says that in its course it gives off sensory fibers to the pericardium and to the pericardial, costal and diaphragmatic pleura, its terminal filaments furnishing sensation to the peritoneum covering the interior surface of the diaphragm.

Irritation or inflammation of this nerve, as in pleurisy, may perhaps be an explanation of some of the cardiac and gastric disturbances, the collapse and meteorism be-

5. Hilton: Rest and Pain, reprint of second edition, p. 252.
6. Van Gehuchten: Systême Nerveux, I, p. 460.

cause the phrenic has communication with the vagus and the splanchnics. The halting movement of the diaphragm, as well as pain referred to the epigastrium and hypochondrium, may likewise be due to phrenic disturbance. Hampeln[7] advocates this view quite strongly, overlooking too much, as it seems to me, the part played by the intercostals in the production of abdominal pain.

It is not necessary to assume in cases of this sort that the inflammation is in the diaphragmatic pleura, i. e., in the part of the pleura anatomically nearest to the abdomen, though undoubtedly this part of the pleura is often inflamed. Rohrer[8] calls attention to the frequent occurrence of inflammation of the diaphragm itself, and believes that to this is due in part the stitch pain, as well as the pain in the abdomen often complained of, especially in children.

Head,[9] who has done so much to explain referred visceral pain, believes that in certain cases there may be abdominal pain when the lung alone is involved without the pleura, e. g., in bronchitis, tuberculosis or bronchopneumonia. He says that through the communicating branches of the seventh, eighth and ninth dorsal nerves, which branches supply the lungs, impulses originating perhaps in a small inflammatory pulmonary focus may be carried up to the posterior root ganglia. From the abdominal wall normal impulses pass from the epigastric and hypochondriac regions to these same ganglia. If, through the abnormal impulse coming from the lung, the ganglia have become disturbed in their function, the normal impulses from the abdomen may be distorted, i. e., misinterpreted, into painful impressions. Head tells Barnard (loc. cit.) that he has seen cases of this sort.

Whatever may be the correct explanation of this abdominal reference of the pain in thoracic disease, the fact that such pain may occur should be clearly recognized

7. Hampeln: Zeitschrift für Klin. Med., xiv, p. 448.
8. Rohrer: Maryland Medical Journal, September, 1902.
9. Head: Brain, 1896. Also Brain, 1900.

by both physician and surgeon. While infrequent, it is more than a curiosity, and it is not so rare that it does not deserve a place in the symptomatology of these diseases.

ILLUSTRATIVE CASES.

I wish by brief reference to cases that have come under my observation, and then by reference to the experience of others, to show that this pain may occur, that it may be a misleading symptom, but that by care it generally permits of a correct interpretation.

In the pneumonia of children abdominal pain is common. The sudden onset with severe pain, fever and vomiting, with the tenderness and distension of the abdomen, make the resemblance to local or general peritonitis striking.

A year and a half ago I was called by two physicians to see a child of three to decide as to the advisability of operation for appendicitis. The history was, as I have stated, sudden abdominal pain, vomiting, high fever, tympanitic and tense abdomen. Yet the cough, expiratory grunt and rapid respiration were plain, as were the physical signs of consolidation of the left lower lobe. Later a pneumococcus empyema developed, healing under drainage.

In this case, as so often happens to us all, the attention had been riveted on the part of the body in which the brusque initial symptoms had appeared, and the later symptoms of trouble in another part of the body, though plain to one new to the case, were thus overlooked.

The lobe involved to produce this pain is not necessarily the lower lobe.

Feb. 22, 1902, I saw with Dr. Klein, a girl of seven years, whose pneumonia began with vomiting, pain and tenderness in the right iliac region, temperature 103, and in whom Dr. Klein found the next morning signs of consolidation of the right upper lobe. A typical pneumonia with high fever, 106, and migration to the middle lobe followed. At the time I saw her the upper and middle lobes alone were solidified.

The same abdominal pain in a left upper lobe pneumonia was present in a 6-year-old child seen with Dr. B. H. Chamberlin, March 12, 1902. The mother described it as "cramps in the bowels." In these upper

lobe cases the explanation of the reference of the pain to the abdomen may be that of Head, of Hampeln, i. e., through the phrenic nerve, or that the pleurisy and accompanying neuritis may be more extensive than the anatomic boundaries of the lobe that is solid, i. e., may involve the lower intercostal nerves.

While in conversation with physicians, I have found some who expressed surprise at, or even unbelief in the onset of pneumonia with sharp abdominal symptoms. I have found many who regard abdominal pain as an important early symptom of pneumonia in children. Some writers on pediatrics are silent regarding that point. Others clearly recognize it, e. g., Baginsky, Henoch, DeMussy and Bouveret. Holt[10] refers explicitly to it in the following words:

> The pain is frequently referred to the loin, the epigastrium or to any region to which the intercostal nerves are distributed. In a recent case, in a boy of seven years, for the first twelve hours there was intense localized pain in the right iliac fossa, associated with such extreme tenderness as to lead to the suspicion that the case was one of appendicitis.

This will be the proper place, I think, to say a few words concerning pleurisy and pneumonia, coincident with or following abdominal disease. Coincident with or following an appendicitis, a pneumonia or pleurisy may appear, the abdominal and thoracic disease both due to the same cause, e. g., the pneumococcus, or the one being secondary to the other. Gussenbauer showed that from thrombosed veins accompanying intestinal obstruction embolic pneumonic foci could appear in the lung. And lately Sonnenburg[11] has fully discussed the similar embolic origin of pneumonia met with during appendicitis, before or after operation. These cases in which the abdominal pain is accompanied by, and probably due to, definite abdominal disease, do not properly belong in the category of those I am considering. But I mention

10. Holt: Diseases of Infancy and Childhood, second edition, 1902, p. 563.

11. Sonnenburg: Lungencomplicationen bei Appendicitis, Archiv. für Klinische Chirurgie, Bd. lxviii, p. 468.

them as in a given case the possibility of the coexistence of both abdominal and thoracic disease must always be kept clearly in mind, a condition not at all infrequent in tuberculosis, carcinoma or general septicemia.

A few months ago I was called to see a girl of about five years, who was regarded as in too weak and desperate a condition to undergo an operation for appendicitis and peritonitis, from which she was said to be suffering. The case was certainly a puzzling one. I was able to demonstrate clearly an extensive consolidation of the lower right lobe and a portion of the lower left. This had not been overlooked by the physicians. The respiration, temperature, mental condition were suitable for pneumonia. But a severe initial and continuous abdominal pain, a rigid, somewhat tympanitic abdomen and a persistent vomiting made the peritonitis seem probable. Yet careful inquiry into the history showed that within 12 hours of the onset the respiration rate had been 52. Palpation also led me to question the existence of a peritonitis, for with care, and by gentle, steady pressure, it seemed to me that there was less tenderness and less real rigidity than were consistent with the supposed extensive peritonitis. This case, illustrated too, I believe, the truth of Barnard's observation that there is a yielding or softening of the abdominal wall at the beginning of each inspiration in these cases where the pain is of thoracic origin. Medicine and food by the stomach being stopped for a few hours, and opium given, the vomiting ceased. There was in a few days a marked improvement in all respects. But convalescence was slow, with some cough, dullness and râles and signs of delayed resolution. No pus was found by a needle in a suspicious area in the right chest. During this convalescence, for a few days the patient had a distinct rigidity of the lower abdominal wall. A complete recovery ensued, the illness lasting altogether about seven or eight weeks.

I am inclined to regard the case as primarily and even solely pneumonic. Yet it is possible that there was here a simultaneous infection of the lung and peritoneum, or the peritoneum may have been involved after the lung, or vice versa. The slow convalescence with persistent abdominal pain was present in one of Richardson's cases.

Turning now to adults, we find that they may exhibit, though less frequently, the same abdominal pain with pleurisy or pneumonia, and this may simulate a peritonitis.

A young man of about twenty, with sudden abdominal pain and fever had some rigidity in the right iliac region.

His physician with whom I saw the case found here a mass concerning whose exact nature he was somewhat in doubt. The possibility of appendicitis was naturally thought of. But he gave a dose of calomel, the mass disappeared, and in twenty-four hours a right lower lobe pneumonia was clearly made out.

I once showed a man in a clinic at the County Hospital with right iliac pain and marked friction rubs over the right chest. He had escaped an operation for supposed appendicitis only because he refused to permit it. Several times patients have been sent to the surgical wards from the examining room with the diagnosis of appendicitis when the more careful ward examination by the surgeon has shown the case to be pneumonia. I know of an instance in Chicago where the abdomen was opened for supposed appendicitis, when the whole trouble was a pneumonia unrecognized at the time of the operation, but which had caused symptoms that were regarded as those of appendicitis. I could cite several other cases in which abdominal pain has been a prominent feature in cases of pleurisy and pneumonia sometimes leading to perplexity as to the correct diagnosis. Thus a woman of 37 years with right pleural effusion told me that her trouble began twenty days before with a pain in the "stomach and side." In another case severe pain just above the crest of the ilium was present for two days before definite physical signs of pneumonia were present.

Dr. Bevan, in October, 1901, operated for me on a patient with acute appendicitis, draining a small abscess due to gangrenous perforation of the appendix. There was a slow but apparently complete recovery. From April to June, 1902, the patient was at her work as a nurse. About June 15, 1902, she had pain in the right chest, temperature 99 to 100. To this she paid little attention, but she became greatly alarmed when she had pain in the old region, i. e., at the site of the scar of the operation. Neither Dr. Bevan nor I could find local evidence for suspecting any recurrence of trouble in the region of the appendix. We thought the pain was referred from the chest in which could be clearly made out a pleural friction and the signs of a small amount of fluid. Within a few days all abdominal symptoms cleared up.

Still another case I may mention. A young lady with a large carbuncle of the upper lip, and with the grave con-

stitutional symptoms attending it—the condition is, as is well known, generally due to the staphylococcus and often fatal—developed a severe pain in the right chest. In the latter location a friction rub with a fine crackling râle and a patch of harsh respiration could be made out. Nothing but the pain over the region of the appendix could be found suggestive of appendicitis, at least to Dr. N. S. Davis, Jr., and myself, who both saw the patient several times, though to some the probability of appendicitis seemed strong. The embolic pneumonic focus was evidently the explanation of the pain. There was ultimate recovery, the later history proving no appendicitis.

Twice I have seen pain referred very accurately to the region of the gall bladder, and the patients in both instances were sure there was cholecystitis, a natural interpretation, perhaps, when we consider that the one patient was a medical student and the other a physician, noted for being poor judges of their own ailments. The student had a right-sided pleurisy. The physician, in addition to the pain, had fever, local tenderness, and was jaundiced. He naturally concluded that he had gallstones and cholecystitis, though the whole trouble was clearly, as the sequel proved, a right lower pneumonia. In this case the pain was pretty definitely located at the point where the tenth rib joins the costal arch, close to the crossing of the parasternal line and the tenth rib, the *bouton diaphragmatique* of some writers. It should be regarded as suggestive of pleurisy, especially of the diaphragm, when this spot is the seat of pain or tenderness upon pressure.[12]

A patient whom I saw with Dr. F. S. Hartmann, was, after nearly two weeks, convalescing from an acute rheumatism and alcoholism when he was seized with a sudden excruciating pain in the upper left quadrant of the abdomen. Dr. Hartmann found him tender to pressure in the left hypochondrium; he soon vomited and became tympanitic. He did not, however, go into collapse immediately after the onset of the pain, and there was no marked perturbation of temperature. Three days later when I saw him, the tympany was less, the vomiting persisted and was grumous, there was an extensive pleurisy to be made out over the left chest, with a dullness and

12. Hampeln: Loc. cit., p. 455. Zulzer: Munch. Med. Wochenschr., 1898, No. 47. Fowler and Godlee: Diseases of the Lung, p. 670.

harsh breathing suggestive of pneumonia. The patient was rapidly failing, and died a few hours later. While no positive diagnosis was ventured by either Dr. Hartmann or myself, we both looked on the pleurisy as probably secondary to some subdiaphragmatic lesion—perhaps hemorrhagic pancreatitis or localized peritonitis from an ulcer of the stomach.

The autopsy made the next morning by Dr. H. G. Wells revealed nothing in the abdomen to explain the symptoms except a high grade of gastritis with submucous ecchymoses. Capillary oozing explained the blood in the vomitus. The left pleura was, however, the seat of an acute inflammation especially well marked over the diaphragm. The lung was edematous and congested with several hemorrhagic areas the size of a hickory nut. The blood showed a pure growth of a bacillus of the colon-typhoid group. Its exact nature was not discovered owing to an accident to the cultures, though the study of the organism as far as carried out made it seem probable that it was the typhoid bacillus.

Here was a case where the diagnosis should read as follows: Acute alcoholism, acute inflammatory rheumatism, typhoid (?) bacteriemia, acute pleurisy involving the diaphragm.

These cases will illustrate, I think, the importance of this occasional symptom of thoracic disease. That my experience is not unique I know from conversation with other physicians, and a few illustrative citations from the writings of others will be convincing on this point. Janeway[13] says he has several times been consulted in cases of pleurisy or pneumonia, where, because of the neuralgic pains reflected over the lower right abdomen, there has been entertained the idea of an operation for appendicitis. Osler[14] says that twice in cases of pneumonia he has seen the suspicion of appendicitis raised because of the sudden acute onset of the pain, once in the region of the navel, and once lower down on the right side. In discussing typhoid perforation, he[15] says: "Abdominal pain of a severe character in typhoid fever may be associated with an acute pleurisy."

---

13. E. G. Janeway: Remarks on Some of the Conditions Simulating Appendicitis and Periappendicular Inflammation. Medical Record, May 26, 1900.

14. Osler: Practice of Medicine, fourth edition, p. 117.

15. Osler: Lancet, Feb. 9, 1901.

This abdominal pain due to pleurisy I once saw in a typhoid. Because of its sudden occurrence and because of a sudden jump in the leucocytes my interne had naturally suspected perforation. Yet an absence of rigidity and of local tenderness and the presence of a fine pleural friction, with harsh localized breath sounds, led me to wait, and the autopsy two days later showed no perforation, but a bronchopneumonia.

Fraentzel[16] saw two cases of pleurisy that closely resembled peritonitis.

Cozzolino[17] refers to four cases reported by Massalongo last year in which cases of abdominal pain in children had aroused suspicion of peritonitis, though the trouble was intrathoracic. Cozzolino's own case was in a child of three years with symptoms like those of appendicitis, including tenderness over McBurney's point, though palpation that was *"dolce, lenta, graduale"* showed little pain and no induration. Next day pneumonia was plain. Guinon[18] also in his case found that gentle pressure with the flat hand and not the finger revealed less tenderness and less resistance than the previous complaint of the patient, a 13-year-old girl, would indicate.

Morse[19] reports three cases in children, one of which was called appendicitis and another gastritis, though pneumonia was in each case, as the later history showed, the cause of the abdominal symptoms. He says: "Within a few years the abdomen has twice been opened in children by well-known Boston surgeons for appendicitis when the trouble was lobar pneumonia."

Brewer[20] reports an operation for appendicitis in a woman 30 years old, the abdominal pain, more or less general ab-

16. Fraentzel: Article on Pleurisy. Ziemssen's Encyclopedia, American Translation, iv., p. 63.

17. Cozzolino: Gazzetta degli Ospedali e delle Cliniche, No. 5, 1903.

18. Guinon: Revue mensuelle des maladies de l'enfance, xix, p. 167.

19. John Lovett Morse: Annals of Gynecology and Pediatry, Nov. 1899, p. 143.

20. George E. Brewer: Some Errors in Diagnosing Conditions Resembling Appendicitis, Annals of Surgery, May, 1901.

dominal tenderness, slight muscular rigidity and distention, with leucocytosis, leading to this diagnosis. The abdomen was opened, but no sign of inflammation was anywhere discovered. The examination of the chest a few days later revealed a circumscribed pneumonia of the right lung, though prior to the operation there were no abnormal chest findings. Two weeks later and after the fever had subsided a small abscess immediately beneath the anterior abdominal wall of the left side, was opened. Brewer thinks that the pneumonic focus had induced the early abdominal symptoms.

He cites another case. A man convalescent from an acute lobar pneumonia, after being free from fever for six days, complained of pain in the right iliac region, vomited and developed fever. As he had years before had what was called appendicitis, a recurrence was naturally suspected. Muscular rigidity and tenderness became marked, the temperature rose, tympanites was extreme and the diagnosis of peritonitis was concurred in by Drs. Janeway, Bull and Brewer, who saw the patient with his physician, Dr. Evans. Because of the desperate condition of the patient an operation was regarded as inadvisable. The autopsy showed a normal peritoneum, a resolving pneumonia of the lower lobe of the right lung, a pneumococcus septicemia. How much of the abdominal pain and other symptoms were due to the pneumonic trouble and how much to the septicemia, it is impossible to say. Yet I cite the case here as an instructive one, and as showing the ease with which peritonitis can be simulated by other conditions.

Barnard's six cases in which abdominal symptoms were prominent, though no abdominal disease was present, deserve to be quoted in full, but lack of time will prevent this. Three were cases of pleuropneumonia involving the base of the lung. In two a pyo-pneumothorax developed, in each case apparently from rupture of a tuberculous focus in the apex of the lower lobe. One was a traumatic pleurisy with broken ribs. In this case the pleurisy was plainly recognized, but so definite were the abdominal signs that the abdomen was opened and found entirely normal.

The most interesting of these cases and one which, as Barnard says, was a most perplexing one, concerned a chlorotic girl of seventeen under treatment for ulcer of the stomach. A sudden violent epigastric pain, vomiting, collapse, rapid pulse, temperature 104.5 F., a tender abdomen, especially in the epigastric region, led to an almost immediate operation for perforated gastric ulcer. Nothing of the sort was found.

On the second day after admission a right basal pneumonia was clearly made out, later also in the left base. The autopsy showed double basal pneumonia and right diaphragmatic pleurisy and "in the stomach a shallow ulcer of the size of a sixpenny piece, which was not even near perforation. There was no peritonitis." Surely this was a hard case to diagnose. As Barnard says, the sudden rise of temperature should have excited suspicion of somethinng else than perforation.

Hampeln's five cases are also worthy of careful study, he dwelling not only upon the possible misleading character of the referred pain, but upon the vomiting, tympany, collapse, etc., that he explains on the theory of disturbance of the phrenic nerve with secondary disorder of the vagus and splanchnics. Surgical intervention in some of these cases was narrowly escaped, and only because Hampeln's experience had led him to examine with unusual care for thoracic trouble. Hampeln's cases were, to quote his own résumé, "two fatal cases of foudroyant pleurisy, one of seropurulent pleurisy with recovery, one fatal and one favorable case of croupous pneumonia, all in the beginning of the illness, creating the impression of a peritonitis or intestinal obstruction, while the ordinary marks of the real disease were absent, and even the physical examination, at least at the beginning, was negative." (loc. cit., p. 450.)

Dr. M. H. Richardson[21] discusses in a very frank manner the possibility of mistaking acute thoracic disease for appendicitis, citing instructive cases. He makes out the error in diagnosis as often due to the fixation of the thought of the observer on the one part of the body or on the one disease. Arguing from a psychologic standpoint, he says:

For a true perception of the normal relation of things to be temporarily suspended, it is only necessary that the mind for the moment be dominated by a fixed idea. Influenced by a single suggestion or by a chain of suggestive circumstances, the mind may become so possessed of a certain idea that the result will necessarily be distorted and incorrect.

---

21. Richardson: Remarks on the Diagnosis Between Acute Appendicitis and Acute Intrathoracic Disease, Boston Medical and Surgical Journal, April 17, 1902, cxlvi, No. 16.

How true this is we all know to our sorrow, in more than single instances. I referred to this point in speaking of the child on whom operation was contemplated for appendicitis. The minds of the two physicians had, as Richardson says, become dominated by the fixed idea of appendicitis, though pneumonia was plainly recognized by both as soon as the possibility of its existence was pointed out. Barnard says the opinion that in his case of traumatic pleurisy there were abdominal complications was largely due to the fact that a few weeks before, with a somewhat similar injury and symptoms, there had been rupture of the spleen with hemorrhage. His mind was drawn, therefore, toward the possibility of a duplicate of this somewhat rare occurrence.

CONCLUSION.

There is little to say in conclusion. Cases may occur in which at first it may be impossible to say whether the trouble is abdominal or thoracic. Each case must be judged on its own merits, and the decision as to immediate operation or waiting, not settled by any hard or fast rule. Certainly in some doubtful cases with threatening abdominal symptoms, it would be better to err on the side of operation and make a laparotomy than to let a possible ruptured appendix, gall bladder, stomach or intestine go untreated for six or twelve hours. An exploratory abdominal incision could, perhaps, be made under local anesthesia or under laughing gas. This would avoid the danger of adding bronchitis, or aspiration pneumonia to an already existing pulmonary inflammation, if such happened to be the primary trouble and the peritoneum is found normal. If peritoneal trouble is found ether or chloroform anesthesia can be added and the reparative surgical procedure instituted.

But with care and circumspection doubt will seldom arise. The main safeguard in the diagnosis is to think of the possibility of a thoracic origin for the abdominal symptoms. It will then be generally found that there is some thoracic pain, as well as abdominal, or there will

be cough or expiratory grunt, perhaps a bloody or rusty expectoration, or the respiration will be increased out of all proportion to the abdominal pain and tympany, or, as in Barnard's case, the temperature will rise too suddenly and too high for the supposed abdominal accident. These facts, if observed, will lead one to a careful examination of the chest, which will, in most instances, even early, reveal some loss of motion, friction, râle, dullness or bronchial breathing that discloses the existence of an intrathoracic inflammatory condition, and makes it clear that the abdominal pain is a reflected one. The abdomen in these cases is often pseudo-tender. A light touch hurts. Quiet, steady, deep palpation with the flat hand does not increase the pain. And, as Barnard observes, at the beginning of inspiration there is a yielding of the abdominal wall that is seldom seen in true peritonitis. With care, then, most of these cases can be recognized. I need not repeat what I said at the beginning, that this recognition is important. That truth is self-evident.

I may add that in this paper I have made no reference to other reflections of the pain of pleurisy and pneumonia, e. g., to the neck or to the lumbar region. Nor have I thought it wise to extend the scope of the paper so as to include such thoracic diseases as pericarditis, aneurisms, tumors, vertebral tuberculosis, etc. I have purposely limited myself to a consideration of the abdominal symptoms, chiefly pain, in acute pleurisy and pneumonia, believing that this is the form oftenest overlooked or misinterpreted.

DISCUSSION.

Dr. J. N. Hall, Denver—There are many points of confusion that I do not think Dr. Herrick has touched. I once saw the daughter of a member of our Association concerning whom two physicians and surgeons were in doubt regarding the presence of appendicitis or pneumonia. It proved to be the former. Confusion existed because there was abdominal distension, the liver was crowded up, and there was dulness below the ninth rib; with the compression of the lung there was

no bronchial respiration, but crackling râles. In such a case it is extremely difficult to say what the condition is.

There is another point of confusion: With the displaced liver we may have friction which can not be attributed to lung or liver exclusively. In other words, it is difficult to determine whether we are dealing with peritonitis or pleurisy. In many cases the pain may be due to a diaphragmatic pleuritis. Again, you may have a leucocytosis, but this does not help in the diagnosis, because it may be due to pneumonia or to appendicitis. Last winter, in the wards of our county hospital, a man was admitted with a diagnosis of appendicitis; on account of the urgency of the case I was asked to see him at once. Everything seemed to be fairly characteristic of appendicitis, and I did not wish to say positively that he did not have appendicitis. I did not recommend operation, and the next day he developed a pneumonia in the left lower lobe and everything subsided in the abdomen. I do not attempt to explain how this happened unless there was a combination of lower left pneumonia and appendicitis.

Dr. Thomas McCrae, Baltimore—Dr. Herrick has pointed out some pitfalls into which all of us may stumble. I have seen three such cases all admitted to the Johns Hopkins Hospital surgical wards. In a recent one there were marked abdominal symptoms which rather suggested an attack of appendicitis, but the patient had had his appendix removed. The appendix being gone, cholecystitis was suggested. Examination showed a very tender abdomen, the pain increasing with respiratory movements. Examination of the thorax revealed slight dulness at the right base. The next day there was tubular breathing. It is easy to be misled in these cases. The sudden onset of abdominal pain with fever, leucocytosis, and, above all, the marked local signs, all suggest an abdominal condition. Inspection may show diminished respiratory movement on one side, there is tenderness, etc. I agree with Dr. Herrick that a knowledge of the possibilities is our greatest safeguard, and with that a thorough and careful routine examination. The surgeons sometimes suggest that we delay too long before calling them in; but perhaps if they called in the physician oftener they would be saved the mortification of opening the abdomen in pneumonia.

Dr. James M. Anders, Philadelphia—It has been long currently stated that the pain is usually located in the vicinity of the nipple of the side affected, but this is shown by statistics to occur only in 50 per cent. of the cases; the pain is often referred to the base of the chest on the side affected. I have seen a number of instances in which the pain was referred to the abdomen. In two instances the symptoms closely simulated those of a cholecystitis, especially the local manifestations at the beginning. Later the pneumonic features

dominated the scene. As Dr. McCrae has stated, the clinical picture very often simulates these abdominal conditions. I can also recall a case in which the pain was referred to the gall-bladder region and in which the patient died, came to autopsy, and a complicating cholecystitis was actually revealed. The diagnosis had been a right-sided pneumonia. During that patient's life the pneumonic symptoms, as is usual, dominated the clinical picture. I can recall another case, seen recently, in which pain was referred directly to the appendicular region. There was considerable rigidity of the right rectus, the abdomen was somewhat swollen, but there was no distinct tumor mass. A diagnosis of appendicitis had been made by two physicians, and it was thought the case ought to be referred to a surgeon for operation. A careful physical exploration of the chest was made, following the golden rule, "in all cases in which there is fever combined with abdominal pain make a careful physical examination of the thorax." A lobar consolidation was revealed. The abdominal symptoms finally disappeared. I believe we may regard the abdominal symptom as an exceptional occurrence after all in pneumonia. In the experience of some the pain seems to be more commonly referred to the lumbar region. It is to be remembered that cases of pneumonia in which the pain is referred to the lumbar region may occur in patients who previously have had a nephrolithiasis. A man aged 62 had nephrolithiasis on two occasions, and more recently a right-sided pneumonia; the pain was referred to the right lumbo-abdominal region. He had marked rigors and a history of a cough, which led to a careful examination of the lungs and the discovery of a right-sided consolidation. The patient insisted that he was suffering from another attack of renal colic on account of the location of the pain. It is most important practically, as Dr. Herrick has suggested, to investigate the thorax before rendering a diagnosis in cases presenting such symptoms as abdominal pains and fever.

Dr. James B. Herrick, Chicago—The diaphragmatic pleura is frequently involved when we have this pain referred to the abdomen; yet it is not necessarily involved. The case that Dr. Hall refers to, where there was a left lower lobe pneumonia, the pain being referred to the right side of the abdomen, is certainly confusing and is not clear of explanation. It is possible there was a slight pleurisy on the right side which was overlooked. It possibly may be attributed to an anastomosis between the terminal filaments of the intercostal nerves of the two sides. Gerhardt and Huss found, in making dissection, an occasional anastomosis between the intercostal nerves of the two sides and explained the pain on the opposite side from this fact. It is possible that some such explanation might explain Dr. Hall's case. Tenderness on pressure is

certainly a confusing feature. Yet, in some cases that I have seen the tenderness has not been exactly that which is found in peritoneal inflammation. If we make gentle steady pressure with the flat of the hand rather than with the finger tips we shall often find that the pain is superficial and not of the kind that is found in peritonitis.

This whole subject is an old one and has been referred to by many writers on pneumonia, but it is frequently overlooked. I did not intend to take a crack at the surgeons, but as Dr. McCrae has opened up this line of thought I may say that in talking on this topic of referred abdominal pain with a couple of surgeons one said he had never heard of such a thing and the other said he did not believe it ever occurred. It was largely because of this conversation that I thought a paper on the subject might be timely and perhaps instructive.

TRAUMATIC PNEUMONIA.

W. T. ENGLISH, A.M., M.D.
PITTSBURG, PA.

The compound term "traumatic pneumonia" implies association of injury with disease. Its ample interpretation concerns alike surgical and medical science. The Greek word "trauma" is very near to that of the English word "injury," both as to use and significance. Pneumonia is a medical appellation given a disease involving the presence of specific germs, and various changes in the air vesicles, parenchyma and associated structures of the lungs. "Pneumonitis" and "inflammation of the lungs," formerly used to designate this malady, have given place to the more liberal term "pneumonia." The use of the latter designation does not commit us to any theory as yet in dispute, and is in harmony with empiric evidences, as well as bacteriologic demonstrations of the present. Under whatsoever garb or name, and whether recorded in the chaos of lapsed eons or mingling with the dust and tumult of twentieth century activity, pneumonia has been and continues to be the assassin of human comfort, health, life and medical theory. Its autobiography is written in signs and symbols so legible that "he who runs may read," without betraying its origin or end. It shows no respect for time, place, age, sex or condition. Now it may be individualistic, again communistic, and anon socialistic. The frontier medical man "sees it in the clouds and hears it in the wind." The bacteriologist, confused by its multo-coloring, announces it an emanation from pleural sources.

Trauma is one causative agency in the awakening of

pneumonia not yet accorded full recognition as a medical or surgical entity, which appeals to the consciousness of the physician of this age as never before. The importance of the subject augments with every step of human advance involving the utilization of energies and mechanical inventions unknown to our forefathers.

With "traumatic pneumonia" the profession is not as familiar as it should be, neither is it equipped with ample literature from those who have been the awed but mute witnesses of its consequences.

Surgical science, as displayed in the war between the north and south, hinted at the subject, and the immortal Hamilton observed that "The shock produced by a musket ball is sufficient to cause a rupture of some of the more superficial pulmonary vessels, and in other cases it gives rise eventually to pleuritis, or even pneumonia." He further says "a missile may impinge upon the thorax and without causing rupture of the skin or anything more than a slight abrasion, may fracture the rib, rupture the lungs, or some of the large blood vessels, or even tear open the heart itself."

Reidinger gave the name of "concussion of the chest," or commotio thoracica, to a series of cases of this character, but produced in various ways which were remarkable for their fatalities.

Litten, a more recent and copious writer upon the subject, recognizes the large influence of trauma upon the occurrence of pneumonia, and concludes that, "lobar, croupous (or fibrinous) pneumonia may arise as a sequel to violence upon the thorax, where no external signs appear." In his terminology he uses the name "traumatic pneumonia" for those cases following penetrating wounds in the lungs, and reserves the term "contusion pneumonia" for those resulting from external force producing concussion of the lungs.

Rezerot notes that "the results of injury may show pneumonia, localized spots of catarrhal pneumonia, and even interstitial thickening of the lungs."

Stephen Paget, a most reliable and capable observer and author, expresses himself thus: "We are tempted to make light of a general injury of the chest, unaccompanied by fracture, or by a serious wound of the soft tissues. But apart from the fact that very grave and even fatal results may follow a blow or a bruise over the chest, which seemed in itself insignificant, general injuries of the chest are sometimes attended by changes so unexpected and so obscure that they need careful consideration." In conclusion, he adds: "We are bound, therefore, not to think lightly of contusions of the chest merely because there is no fracture or external wound."

Pirogoff is authority for the statement that "such injuries are at least as dangerous as penetrating wounds."

Stern, who has made a special study of pneumonias from all causes, places the statistics of those produced by trauma at 4.4 per cent.

From all this we must infer the importance of the subject presented, and be also impressed with the necessity for careful inquiry into the causal factors in all classes of pneumonia, and especially those preceded by injury, no matter how trivial the objective evidences of trauma may appear. We must not forget that accident and fright may be factors in the production of traumatic pneumonia. Some recent authorities claim that fright is the first element of danger, although manifestly greater in some than in others. In our study of the factors contributing to the occurrence of traumatic pneumonia, we need not prove faithless to the theory associating the disease with the diplococcus, as it is a fact that the micro-organisms are present in the expectorations of those suffering from traumatic pneumonia.

The purpose of this paper is the consideration of those cases of traumatic pneumonia which appear from con-

tusions, and, as far as possible, refrain from mention of those due to penetrating injuries of the lungs, or those including fractures of the bones or cartilages.

The question arises, "how is it possible for a jar or force expended upon the external surface of the yielding thorax to be so transmitted as to do violence to the contained viscera, and especially the elastic lungs?" We may understand why the delicate tissues of the brain may be injured by slight concussion, even by contrecoup or force upon the lower end of the spine, but we are naturally inquisitive about like possibilities in the lungs. The normal elasticity of the chest, in the majority of cases, is capable of rendering *nil* the accidental forces which antagonize it. The extremely young will resist a crushing weight, as that of the wheels of a wagon, to which the mature thorax would yield. However, a stroke of 100 to 300 pounds, carrying with it past the point of contact upon the chest all of the force not antagonized by the resistance offered, will cause the elastic walls to impinge upon the lungs with dangerous results, corresponding to the degree of thoracic elasticity. It would be more to the pulmonary disadvantage if the lungs were surprised by accident during full inspiration than if occurring at the end of expiration. Unfortunately, the former is the attitude of the lungs at the instant when the largest number of injuries are received. Fright is associated with 90 per cent. of chest injuries. In more than half of these fright prompts a gulp of air, closure of the glottis, rigidity of the diaphragm and other muscles of respiration and a fixidity of the thorax. There is also an attempt to free the arms, as in the attitude of self-defense. The chest is never guarded in this pose, instinctively responsive to the primal law of nature—self-preservation. Fright has not time for appeals to the ideational centers. The frightened human "holds his breath," and a stroke or fall on the chest is often received in the position of pulmonary disadvantage. To this condition of the chest is often added an advan-

tageous angle for the force because of the unfortunate position assumed by the individual.

Hamilton describes the mechanism of force thus: "The ribs with their cartilages are bent inward violently and made to encroach very far upon the contained viscera, and serious lesions may be produced before the bony parietes are fractured."

Thus far we have considered normal conditions of the thorax and lungs, and the inference is that the force required to produce injury to the lungs varies with the age, elasticity of the chest, position of the lungs in the respiratory act, the exact angle at which the stroke may be received, and the individual factors.

It must be remembered that if a blow impinges upon the chest walls it is distributed to adjacent structures much as the sound waves in the air, amplifying and making greater impressions upon localities most easily influenced.

Peyrot claims that if a blow is received by the chest it is distributed to adjacent structures, but the lung itself is independent of this movement. Any want of elasticity, however, will favor yielding under stress, and injury results. This is direct injury. Moreover, there are numerous foci of latent weaknesses and chronic maladies in the average lungs which are liable to be affected by force thus extended. Tubercular diseases, pulmonary emphysema and congestions, as from heart disease, alcoholism, etc., act as predisposing causes and give wider range to traumatic influences than would be available in normal conditions.

Adhesions may influence changes in a mechanical way in parts remote from the seat of force. The jar may awaken a latent disease protected hitherto by areas of inactivity and solidification.

Many intrinsic and extrinsic causes must be considered in the efforts to account for pneumonia which will appear peculiar to each case and constitute the individual factors. One thought must be ever present with

the physician called to examine the chest after injuries, and that is the universal possibility afforded everywhere for acquiring traumatic pneumonia. Pneumonia is today made available to all. The wholesome and the unwholesome, the wise and the unwise, are almost equally liable to become its victims. Nor is it needful that they await the incubation period of the pneumococcus or streptococcus, but by thoughtless dalliance during the millionth part of an instant in the way of a rapid transit electric car or a twentieth century juggernaut—the automobile—pneumonia begins to weave the network of death in the lungs. From the cultivated "interference" of the foot ball field, the polite fistic arena and other places for the display of athletic proficiency, as well as amid the hum and whir of mill and workshop, and from every point of the world where the strenuous life of the twentieth century thrills human kind, traumatic pneumonias are reported. These augment the numerical statistics of pneumonia and increase the mortality.

Interest in the subject is enlarged also by its medicolegal aspect. It is required that the physician commit himself most positively and under oath to the theory of its cause and consequences. The numerous incidental notations which concern the history, the onset, complications and sequelæ will be investigated by life assurance companies, accident insurance associations, and other corporations or individuals charged with liability, and tedious trials and litigations demand of the attending physician the most careful, exact and conscientious judgment of which he is capable.

The question will arise, "is the pneumonia an accidental or coincidental circumstance?" This must be answered by the physician in every case involving medical and surgical possibilities, whether before the legal judiciaries or the bar of his own conscience.

The responsibility of the physician ends not with the apparent cure of the acute conditions, but continues

without limit because of diseases which develop later and may be regarded as sequelæ.

Pleurisy, pneumothorax, pyothorax, empyema, phthisis and almost every malady in the category of chest disorders have been charged to injury received at some period in life. The postmortem evidences have been most effective in some investigations.

When a physician is called to a case of injury liable to produce traumatic pneumonia he will probably find a rapid pulse and accelerated respiration, and in fully 65 per cent. the sputum will show some blood within a few hours, if not immediately. The shock is the first element of alarm demanding attention. The respiration reaches 60 to 70 per minute, but is not so common a symptom in adults as in children, and does not always correspond with the extent or gravity of the injury. Hamilton notes that "expectorations of blood in most cases occur immediately—frequently not until after the lapse of several hours, or even days, but it is rarely seen after the fifth, sixth or tenth day." The writer observed in a recent case that spitting of blood continued until the fourteenth day after the injury. Dyspnea may be absent, and no duskiness of the face, and nothing to suggest injury except disturbed breathing. No general or extensive contusion may appear with the history of a slight blow, affecting one portion of the chest and yet the shock be most disastrous. A condition of syncope immediately following trauma may be due to direct stimulation of the vagi, or to direct cardiac compression. The depressor nerves and the sympathetic may also act as causative agents in lowering the blood pressure during any prolonged period.

The examination of these cases must be conducted with celerity, and the physician should endeavor to give the patient as little discomfort as possible. The horizontal position is usually the safest and one causing least suffering. The external bruise or contusion will heal rapidly, and the pneumonia usually gives its character-

istic signs and symptoms after forty-eight hours from the time of the contusion. This pneumonia may or may not be promoted by a destruction of continuity of the lungs, pleura or bronchial tubes, and the mechanical jar of the impinging stroke cause a slight tenderness or soreness which may arouse latent pneumonic energies which otherwise might have continued in abeyance or be counteracted without reaching the dignity of a pneumonia. Slight concussions are rarely dangerous in a purely surgical sense, but can thus directly prove fatal, and it is well to remember that infection may come to the lungs thus crippled and phthisis speedily follow. The relations of time to these consequences are very variable, and it is not safe to say that pneumonia coming on a fortnight after the injury of the lungs is not due to concussion, nor is it to be regarded as a safe position to assume that a phthisis, following years after the lungs have shown apparent cure, bears no responsibility as a causative agency. The rusty sputum is likely to succeed upon the streaked sputum as soon as the pneumonia reaches its second stage. Moreover, the diplococcus is to be found in traumatic pneumonias as easily as in cases free from injury. It is a fact that these germs are to be found in the mouths of most healthy people.

Jobert separates into three degrees, or classes, these injuries:

First—Hemorrhagic punctata from capillary rupture, the parenchyma remaining intact.

Second—Includes those in which small ruptures exist in tissue, including alveoli, bronchioles and vessels about them.

Third—Tears of the lungs, bronchi and vessels.

No systematic or conventional method of observation or treatment can be provided for these cases, as each is a law unto itself, yet a review of a few of the records may prove of advantage.

E. Block tells of a man 41 years old falling and strik-

ing upon the lower portion of the right side of his chest, who developed signs of pneumonia the following day in the right lower lobe. No chill was recorded, and the crisis occurred at the end of the fourth day. Bloody sputum was absent. It is assumed in the report that a moderate injury of the lungs may have given opportunity for the invasion of the pneumococci, which were found in the sputum.

Dr. R. A. Booth, physician to the City of Pittsburg, is quoted as having a case of traumatic pneumonia a few days ago in the hospital, caused in a man 62 years old, and which proved fatal in the fourth day. At the hospital the patient told the attendants he was injured by falling down stairs. A number of cases gathered by research have been charged to similar accidents.

R. E. Lord has placed on the list a case of injury to the chest by striking against a desk followed by hemoptysis and consolidation of the lung immediately underlying the seat of injury. This is one of a number of reports of cases of traumatic pneumonia resulting from violent contact of the chest and the edges of desks.

Dr. A. Koch adds the case of a man aged 38 years who fell from a height and received a severe contusion of the left side of the thorax, and in less than forty-eight hours after died in the hospital. Postmortem examination showed croupous pneumonia, with red hepatization and pneumonia bacilli. The man was free from disease at the time of the accident, and the ribs were not injured.

A case of "contusion pneumonia" contributed by Aufrecht shows that the pneumonia occurred on the side opposite to the injury, which later was produced while pushing a wheelbarrow heavily loaded, when the left handle struck upon the left side of the chest. The man was 38 years old, and up to the time healthy. About ten hours after the accident pneumonia appeared on the right side. Three days after the injury he died. An autopsy showed pneumonia of the right base.

During a professional experience of over three decades there have been personal opportunities afforded for observation in the City of Iron and Smoke. The list of traumatic pneumonia, inclusive of fractures of the bones and cartilages, as well as those due to contusions and concussion, challenges credulity. Those within the scope of this paper are not so numerous, and a brief recitation of some of these will be interesting. A man aged 29, of good health and uncertain family history was thrown upon his back and rudely tossed against the front door of an electric car which had deserted the track at a sharp curve and collided with a telegraph pole. He was completely disabled, and was lifted into a carriage and conveyed to his home, a distance of five blocks. Dyspnea, with the psychologic evidence of excessive alarm, were associated with blood-spitting and complaints of unease within the thorax. Appropriate treatment speedily annulled the immediate signs of the shock. Perfect quiet in the recumbent position was secured, but notwithstanding these precautions, a traumatic pneumonia was in evidence before the conclusion of the first forty-eight hours, without any special features, except that the hemoptysis gave place to rosy sputum on the third day. This case terminated by lysis, and was followed by prolonged convalescence. Three months later hectic symptoms appeared and tubercle bacilli were found in the sputum. A few days short of two years after the accident the life terminated. A suit was brought by the relatives against the street railway company, whose legal advisor made satisfactory settlement.

A man aged 61, of reasonable health, who was filling the duties of labor boss, was caught between an overturned wagon and the side of an excavation. His release was affected after about eight minutes. He was faint and suffered from a sense of suffocation. The employment of alcoholic stimulants, administered by the mouth, and crude attempts at artificial respiration gave him some relief until medical aid arrived. Hypodermatic

use of strychnia and brandy were resorted to with good results. He was removed to his home and placed in the most restful and quiescent condition possible. This man developed a pneumonia before the end of the first twenty-four hours, in which the temperature range never exceeded 103.8. The pulse, which was 148 during the period of shock, fell to 118 after the lapse of ten hours, but fluctuated between this and 138 without apparent cause. This case showed no real abatement until after the fourteenth day, when the pneumonia ended rather abruptly, and all symptoms disappeared gradually. The patient resumed his former occupation at the beginning of the sixth week.

A man aged 57 years was walking on the railroad track when he was struck by an engine and precipitated over an embankment of 16 feet, sustaining fractures of the right humerus in two places. The force of the stroke caused the arm to infringe upon the right side of the thorax, producing an extensive contusion over the entire side. Blood-spitting appeared immediately. After sixteen hours, and without chill, the temperature reached 103.8, respiration 44 and pulse 128. The signs and symptoms of pneumonia continued until the seventh day, when they suddenly diminished and convalescence was rapid, leaving only the external surgical conditions for treatment. After a lapse of twenty-eight months this man continues in perfect health.

Dr. Ulysses Strouss of Beaver, Pa., reports a case in a man aged 32, standing on the rear end of his wagon unloading coal, when his horse suddenly started forward and he was thrown violently to the pavement, alighting on the posterior superior portion of the chest. He spat blood in a few minutes after the accident, which was succeeded by traumatic pneumonia, terminating by crisis on the fifth day, and there was rapid recovery. Six months have elapsed and no unfavorable symptoms have appeared.

A case illustrative of the lapse of time before betrayal of the results of injury is that of a man riding in an

automobile and colliding with an electric car, precipitating him to the pavement with considerable force, upon the posterior aspect of the chest. The injury did not retire the subject from his duties until after the expiration of ten days, when a large quantity of dark blood was expectorated, in which the molds of the bronchial tubes were plainly demonstrated. The temperature ranged during four days between 102 and 103, when it ended by lysis. The physical signs were those of bronchopneumonia, with copious mucous râles of varied size, and cough and respiration of suffocative character. The sputum was examined carefully for tubercle bacilli with negative results. Subsequently, by the city physician, with like report. A third specimen expectorated in the writer's presence, and divided into three parts, was examined by three different persons, and several bacilli discovered by one. The last reports have been that the patient is perfectly well. This accident occurred ten months ago.

A man aged 41, lumber dealer, and of good habits and health, was thrown from the steps of a rapid transit car while attempting to alight, the signal to start being premature, and he received a severe stroke upon the anterior portion of the chest walls. He was taken to his hotel and placed in bed. No fracture existed, but in a few minutes after the fall he expectorated some blood-streaked sputum. Ten hours after the accident a fever of 104, pulse 138 and respiration of 52 were observed. The sputum became thoroughly mixed with blood and rusty. This case was prolonged, ending in lysis the fourteenth day after the advent of fever. Convalescence was slow, and the patient never recovered fully from a bronchial catarrhal condition, with numerous streptococci, but no tubercle bacilli in sputum. This case is still under observation. The chest is of paralytic appearance and conformation, and the subject admits the history of former bronchial catarrh, but claims that it was but temporary.

One of the most remarkable cases in the writer's experience was that of a man aged 31 years, 5 feet 8 inches in height, weight 168 pounds, of good family history and excellent health, a puddler by occupation, who was struck forcibly upon the right anterior portion of the chest by the handle of the tongs with which he was transferring a ball of metal. He immediately became faint and was seized with dyspnea. A physician examined the chest without discovering any external or internal signs of injury, except a swiftness of respiration. After the use of hypodermics of strychnia, brandy and other antidotes to the shock, he was able to go to his home by street car, a distance of about four miles. Here he received attention during the succeeding four days, and the diagnosis was pneumonia. The temperature, pulse and respiration, rusty sputum, etc., were in evidence in the history, as related to the writer, who was called in consultation on the fifth day, because of the alarming dyspnea which suddenly appeared. The patient was on his knees on the floor pressing the anterior portion of the chest against the mattress and begging for relief for his breathlessness. His features mirrored his desperate suffering. The side opposite to the pneumonia was distended to its utmost by pneumothorax. A paroxysm of severe coughing had preceded the pneumothorax, and doubtless produced a bronchial communication with the pleural cavity belonging to the lung engaged in supplementary respiration. The discomfort was reduced by a hypodermic injection of morphia gr. ¼, repeated in two hours. The proposition to use a trocar and give exit to the air was persistently rejected. After twelve hours it was determined to give surgical relief, regardless of the patient's opposition. At the hour appointed for the operation the patient was lying calmly in bed, the signs of distress had departed, respirations were reduced from 56 to 40, the pulse from 132 to 110, and the temperature had fallen 3.8 degrees. The physical signs of pneumonia were not gone, neither were

those of pneumothorax wanting, but to each side they were much reduced, and the operation was abandoned, and the case progressed to a favorable termination of the pneumonia, which had ended by crisis, but the evidence of effusion succeeded the pneumothorax. The patient, notwithstanding all this, gained flesh, refused to have the fluid removed, and worked six months at the puddling furnace, after which he visited his home in Scotland, returning to his duties at the end of four months. He presented himself for examination one year and two months following the accident with a pyopneumothorax which had evacuated itself of one pint to three pints of pus through the mouth, repeating the evacuations at intervals of twelve to sixteen days. Specimens of this fluid examined on several occasions revealed numerous tubercle bacilli, streptococci and other microorganisms. This spontaneous method of relief continued during seventeen months, when it ceased, and the pleural cavity became distended, to finally present an empyema necessitating pointing between the ninth and tenth ribs on the vertical midscapular line. A hectic condition continued the pulse, temperature and respiration elevations for some time.

He was taken to the hospital and the chest opened, drained and washed out and treated with antiseptics. After three weeks he returned to his home much improved, but gradually yielded to phthisis eight months thereafter.

The study of these cases must include their medical, surgical and legal aspects. This has been advanced by references to numerous authorities and cases. Diagnosis, differentiation and treatment are so interwoven that it seems like supererogation to go into any further detail. The prognosis has been intimated. The medical treatment should be similar to that of ordinary pneumonia plus the demands of the surgical contributions. Complications or sequelæ are also subject to general and well-known medical and surgical management.

THE TRANSMISSION OF YELLOW FEVER.

JAMES CARROLL, M.D.
ASSISTANT SURGEON U. S. ARMY.

Several American observers have noted that during the yellow fever outbreaks mosquitoes were very numerous, among them Rush,[1] at Philadelphia in 1797; Dr. Weightman,[2] of the U. S. Army, at St. Augustine, Fla., in 1839; Dr. Wood,[3] at Centerville, Miss., in 1853. Dr. E. H. Barton[3] states "that at Clinton, La., in 1853, mosquitoes were uncommonly numerous night and day." At Trinity, La., in the same year, when sawdust was used to fill up low places in the streets, the disease was not propagated, though many cases were brought there and no precautions were used.[3] It is hardly necessary to note what the effect of this obliteration of puddles would be on the development of mosquitoes. Dr. Beyrenheidt[3] of Biloxi, Miss., in reporting a severe epidemic at that place in 1853, during which 533 cases and 111 deaths occurred in a population of 5,520, makes the interesting statement that "mosquitoes and fleas were very abundant." Dr. Bennett Dowler strongly urged drainage of the city of New Orleans, and spoke of the city and its environs as "mosquito lands."[4] La Roche[5] states that

1. Medical Inquiries and Observations, Benjamin Rush, M.D., Philadelphia, 1805, vol. iv., pp. 10, 77, 175.
2. An Essay on Yellow Fever, by B. B. Strobel, M.D., Charleston, S. C., 1840, p. 162.
3. Report of Sanitary Commission of New Orleans on the Epidemic of Yellow Fever of 1853, New Orleans, 1854, vol. i., pp. 266. 278, 540.
4. The Epidemics of New Orleans, by Bennett Dowler, New Orleans, 1854, p. 44.
5. La Roche on Yellow Fever, Philadelphia, 1855, vol. i., pp. 67, 77, 79.

in 1793, during the epidemic in Philadelphia, "the narrow streets and alleys near the wharves, as, indeed, in many other parts, were in a foul state, and the gutters almost everywhere sadly neglected." In 1794 Rush and others ascribed the fever to exhalations from wharves, neglected gutters and stagnant ponds.[5] In 1797, during another outbreak in Philadelphia, the condition was practically the same; the Academy of Medicine called the attention of the governor to the putrid exhalations from the gutters, streets, ponds and marshy grounds of the neighborhood of the city.[5] While high temperature, low altitude and the presence of standing water are the conditions most favorable to the development of a yellow fever epidemic, there are numerous instances on record where the disease after introduction spread from house to house throughout the town in places where the general conditions of sanitation and drainage were good. This is now explained by the habits of the mosquito (*Stegomyia fasciata*), which breeds in standing water, in houses as well as out of doors. As a general rule, in ports where yellow fever epidemics first appear, the conditions are known to be favorable for the development of enormous numbers of mosquitoes.

A very interesting observation was made in 1839 in South Africa by Wm. Ferguson, then a surgeon in the South African corps, in regard to the extension of yellow fever from Sierra Leone (where it had been brought on vessels from the West Indies) to Gambia, Goree and Ascension, the subsequent ports of arrival of some of these infected vessels. After mentioning an unusually heavy accumulation of mud and filth in a pit at Ascension, which was said to have been the cause of the fever occurring after an unusually heavy fall of rain, he makes the following statement:[6] "It will be observed that at Ascension, at Goree and at Gambia, a period of three or four weeks always elapsed betwixt the landing of the sick

6. See footnote 2, p. 87, citing from the London Medical Gazette for August, 1839.

and the epidemic outbreaking of the disease among the population, a degree of uniformity worthy of remark, whether the conclusions at which I have arrived (that the disease was carried to these places on infected vessels) be mistaken or not." These outbreaks were accompanied by black vomit and were undoubtedly yellow fever of imported origin.

Just sixty years later Carter[7] made a similar observation in the United States, shortening the period slightly and giving cases and data. In a later paper[8] he designates the interval between primary and secondary cases the period of "extrinsic incubation."

The known facts regarding the apparent non-contagiousness of the disease, the peculiar manner in which it has been seen to pass from one house to another without intercommunication of the inhabitants, and the numerous points of resemblance between yellow fever and malaria, led us to expect results from mosquito inoculations in yellow fever. After satisfying ourselves that *B. icteroides* (Sanarelli) was not to be considered, Drs. Reed, Lazear and myself, in consultation, decided that the mosquito theory had many facts to support it and could be promptly decided one way or the other. This leads us to a consideration of the mosquito theory of Finlay.

At the session of the Royal Academy of Sciences, Havana, Aug. 14, 1881, Finlay first gave to the world his memorable theory of the transmission of yellow fever by the mosquito, and reported five experiments, which are included in the accompanying table, comprising all of Finlay's and Delgado's positive cases published in 1891.[9]

Finlay believed[10] that the transfer of the specific agent

7. New Orleans Med. and Surg. Jour., May, 1900.

8. Medical Record, N. Y., June 15, 1901.

9. Anales de la Academia de Ciencias Medicas de la Habana, Havana, xxvii, 1890-91, p. 501. (Finlay.)

10. Original Article—republished in English in the Revista de la Asociacion Medico-Farmaceutica, Havana, Cuba, Jan. and Feb., 1902. (Finlay.)

of the disease from patient to non-immune was effected mechanically by the insect's proboscis,[14] and that the infection would be conveyed immediately if the insect chanced to bite a susceptible person a few moments after feeding on a patient in the proper stage of the disease.[11] He also thought that the graver forms of yellow fever might be produced by the bite of *Culex pungens,* because the insect ingested a larger amount of blood than stegomyia, and consequently a greater quantity of the virus would be retained on its proboscis (stylet).[11] He ventured the opinion that yellow fever was a sort of eruptive fever, and that the seat of the eruption was the endothelial lining of the blood vessels, and that the mosquito picked up the inoculable material from the interior of the blood vessels on its biting apparatus and transferred the same to the interior of the vessels of the person subsequently bitten.

In a later paper he assumes that the picking up of these germs by the proboscis of the mosquito is selective, because there is nothing to prove that other inoculable diseases have ever been so transmitted. From four culture experiments, in each of which the head and proboscis of a mosquito was dropped into a bouillon tube, he infers that the head and proboscis of the day mosquito possesses bactericidal properties for ordinary bacteria and fungi with the exception of a coccus, which was subsequently[12] claimed to be the specific agent of the disease.[13] The sting of the mosquito is suggested as the "intermediate host" necessary for some phase of development of disease germs.[14]

As late as 1899 Finlay mentions the isolation of the tetracoccus from culture media into which the heads and proboscides of contaminated mosquitoes had been

11. Finlay: Ibid., pp. 362 and 369; also Edinburgh Med. Jour., Oct., 1894, p. 334.

12. Proceedings of the International Congress of Hygiene and Demography, Budapest, 1894, p. 702.

13. Edinburgh Med. Jour., vol. xii, 1, pp. 523 to 526.

14. Finlay: Amer. Jour. Med. Sci., n. s., vol. xcii, 1886, pp. 402, 403.

TABLE 1.—MOSQUITO INOCULATIONS REPORTED BY FINLAY AND DELGADO IN THE ARCHIVES DE MEDICINE NAVALE, TOME 55, PARIS, 1891.

No. of Case.	No. of Patient.	Date of Contamination of Mosquito.	Date when the Patient was Bitten.	Period in Mosquito.	Date of Attack of Fever.	Period of Incubation.	Character of Attack.	Albuminuria?	REMARKS.
1	F. B. M. .	June 28, 1881 .	June 30, 1881 .	2 days	July 14, 1881. .	14 days	Mild . . .	Yes . .	Excessive period of incubation.
2	Aly. C. . .	July 16, 1881. .	July 22, 1881 . .	6 "	July 27, 1881 . .	5 "	Abortive .	No . .	Insect bit Dr. Finlay July 20.
3	L. G. P. .	July 26, 1881 . .	July 29, 1881 . .	3 "	July 31, 1881 . .	2 "	Mild. . . .	No . .	Infection by mosquito excrement.
4	D. L. F. .	July 29, 1881 . .	July 31, 1881 . .	2 "	Aug. 5, 1881 . .	5 "	Abortive .	?. . . .	Did not report himself sick. History of illness vague.
5	D. G. B. .	July 31, 1881 . .	Aug. 2, 1881 . .	2 "	About Aug. 20, '81	18 "	Mild . . .	?. . . .	Excessive incubation period.[15]
8	J. B. . . .	June 20, 1883 .	June 22, 1883. .	2 "	July 9, 1883 . .	17 "	. . . . .	No . .	Two mosquitoes used. Excessive incubation period.
								No . .	Resided in Havana.
9	P. U. . . .	Aug. 13 & 15. '83	Aug. 17, 1883 . .	. . .	Aug. 26, 1883 . .	9 "	. . . . .		Excessive period of incubation. Jesuit priest, lived at
								No . .	Marianao near Havana.
13	J. S	. . . . .	Aug. 18, 1883 . .	. . .	Sept. 9, 1883 . .	22 "	. . . . .		Excessive period of incubation. Lived at Marianao.
								No . .	Moved to Havana six days before his attack.
28	H. Oz. . . .	. . . . .	Sept 7, 1886 . .	. . .	Sept. 23, 1886 .	16 "	Mild . . .		Excessive period of incubation.
32	J. P. . . .	. . . . .	June 16, 1887 .	. . .	July 11, 1887 .	25 "	. . . . .	Trace..	Excessive period of incubation.
42	P. Rem . .	. . . . .	June 3, 1888. .	. . .	July 1, 1888 . .	23 "	. . . . .	No . .	Excessive period of incubation.
55	P. Go . . .	Not stated . .	Aug. 17, 1889. .	. . .	Aug. 26, 1889. .	9 "	. . . . .	No .	Excessive period of incubation.
66	H. Ola. . .	Not stated .	Aug. 14, 1890 . .	. . .	Aug. 21, 1890. .	7 "	. . . . .	No . .	Lived at a country residence near Havana.

dropped, as one of the three results of his experiments on yellow fever mosquitoes.

In a paper[18] published in 1895 he announced some very interesting results obtained with the "tetracoccus." A number of rabbits were inoculated intraperitoneally with cultures in bouillon; one of these cultures had been obtained from the head and proboscis of a mosquito by placing them in a tube of bouillon five days after the insect had been a yellow-fever patient; the others came from the finger blood and milk of yellow fever patients. All of the rabbits died; some of them showed infarcts of the kidneys, lungs and liver. A rabbit obtained from the country was placed in the stable where some of the other rabbits had been kept, and at the end of eight days it was found dead. The tetracoccus was obtained from its heart's blood.

Eleven days after the rabbits had been removed from the house (the stable is usually a part of the house in Havana) a Spanish wet nurse came to spend some time there, and on the third day following her arrival she was attacked with yellow fever. Mosquitoes had been unusually abundant in the house during the whole time the inoculated rabbits had been kept in it, and we are asked to keep this in mind, in view of the possible transmission of the disease from animals to man. We are told that the blood from the finger of this woman on the third day of her illness gave the "pale yellow tetracoccus," and the milk from her breast gave a "white tetracoccus," which proved fatal to a rabbit in fifteen days.[18]

While reading the accounts of these experiments one is constantly reminded of the pathogenic staphylococci. It is strongly suggested[18] that the rabbits died of yellow fever, and it is asserted that the experiments justified the assumption that the tetracoccus was the "specific germ of yellow fever."

The statement is also made in this paper that of one hundred presumably susceptible persons who had re-

ceived the mosquito inoculation during the fourteen years previous, only three had subsequently died of yellow fever.[13]

I have been unable to find records of these one hundred cases in the library of the Surgeon-General's office, and submit those of which the data are accessible to me. As these include the cases on which Dr. Finlay's claim is based they will suffice for the purpose. The fifty-four negative cases are omitted.

Case 3 was bitten by a mosquito that had been fed on bloody excrement from another mosquito that had been found inside the mosquito net of a fatal case. The material was allowed to dry for twenty-seven days in the test tube and was then moistened with a little sugar and water to prepare it for feeding.[15] There is a marked difference between the method of procedure herein described and that given in 1901,[16] when Dr. Finlay said, "I applied a nocturnal mosquito and allowed it to fill itself from a case of yellow fever. After pricking the side of the insect's abdomen I mixed the blood which escaped with a drop of sterilized syrup and fed with it a fresh culex mosquito, and finally applied the latter to a third soldier (July 29). Two days later July 31, the soldier went to the hospital with a fever which was also recorded as "abortive yellow fever." This case (L. G. P.) was omitted from the paper published in 1881, as stated, but was included in 1891 in the report from which the cases here recorded are taken.

Case 4 is further discussed in English in a foot note on p. 368 of the Revista de la Asociacion Medico-Farmaceutica, Havana, February, 1902. (History of illness vague. Did not go to bed or report himself sick.) The first five cases were newly-arrived soldiers, stationed at Cabanas and who were brought to the city of Havana for examination once in every five days.

Case 9 is cited by Finlay[17] in refutation of our conclusion[18] that the mosquito is not capable of infecting until about twelve days or more have elapsed after it has bitten the patient.

Case 55 was a Spaniard, newly arrived, who lived at a country residence. At both attempts to inoculate him the mosquitoes failed to bite; nevertheless he was taken sick August 26. Another non-immune who occupied an adjoining room was taken sick with similar symptoms, a few days later. No further comment is necessary, except that the cases of the

15. Footnote to p. 369, Revista de la Asociacion Medico-Farmaceutica, Havana, Feb., 1902.

16. Medical Record, N. Y., Feb. 9, 1901, p. 203.

17. THE JOURNAL A. M. A., April 13, 1901, p. 1041.

18. Ibid., Feb. 16, 1901, p. 439; 3d conclusion.

Army Commission and of Dr. Guiteras were kept under close observation and strict control. Nearly all of Dr. Finlay's cases were exposed to infection in the city of Havana. Only four of them come within the recognized periods of incubation, viz., Nos. 2, 3, 4 and 66. The first three of these were inoculated in Havana in July, 1881, during which month there were ninety deaths from yellow fever in that city alone. The last was inoculated in August, 1890. In that month sixty deaths from yellow fever are recorded for the city of Havana.

Finlay has published a great many conjectures and speculations in regard to yellow fever transmission. Some of these, such as the belief that the disease was transmitted by the mosquito, have been proven by our work, but not in accordance with his ideas. I can find no evidence that he has produced a single case of yellow fever by his inoculations. In the first place the mosquito is incapable of transmitting the disease in so short a period as two to five days after biting the patient. In the second place seven days must be placed as the extreme limit of incubation in the human being. Our longest period was between five and six days. Finlay conveniently puts the ordinary limit of yellow fever incubation between five and twenty-two days.[14] The efficiency of the five-day quarantine regulation proves that incubation in man certainly does not extend over six days, or seven at the extreme, and in the thirty experimental cases recorded by ourselves and Guiteras the former period has never been exceeded.

In a letter of Aug. 20, 1901, Dr. Finlay[19] states he was convinced that extension of the interval between the application of the mosquito to the patient, and subsequently to the non-immune, would develop a severe attack, such as he was anxious to avoid. One can sympathize with the humane feeling that prompted him to forego positive proof of his theory rather than run the risk of producing a severe attack, but it is a fact that on Aug. 22, 1901, two days later than the date of that letter, I saw Dr. Finlay apply to a non-immune two

19. Medical Record, Aug. 31, 1901, p. 844.

mosquitoes that had been kept thirty-four days after contamination.[20]

I have been impelled to look up the literature of this subject because I have seen several times of late the statement of Dr. Finlay that the Army Yellow Fever Commission has tacitly ignored his right of priority.[21] It is

TABLE 2.—NEGATIVE MOSQUITO INOCULATIONS OF THE ARMY COMMISSION.

Number.	Character of Attack and Number of Patients Bitten.	Day of Disease.	Interval in Mosquito. (Room temp.)	Date of Application of Mosquito.	Number of Mosquitoes Applied.	Remarks.
1	Mild, 1	7th. .	5 days... .	Aug.11, 1900	1	Negative.
2	Very mild, 1. .	5th. .	5 " . .	" 11, 1900	1	"
3	Very mild, 1. .	5th. .	6 " . .	" 12, 1901	1	"
4	Very mild, 1. .	5th. .	6 " . .	" 12, 1901	1	"
5	Very mild, 1. .	5th. .	8 " . .	" 14, 1901	1	"
6	Very mild, 1. .	5th. .	10 " . .	" 16, 1901	1	"
7	Severe, 1 . . .	2d. .	3 " . .	" 18, 1901	1	"
8	Very mild, 1 .	5th .	13 " .	" 19, 1901	2	"
	Severe, 1 . . .	1st. .	3 " .			
9	Fatal, 1.	2d. .	6 " .	" 25, 1901	1	"
	Mild, 1	1st. .	4 " .			
	Severe, 1 . . .	2d. .	2 " .			
10	Severe, 1 . . .	3d. .	3 " .	Nov.20, 1901	1	" Repetition of No. 10.
	Severe, 1 . . .	3d. .	6 " .			
	Severe, 1 . . .	5th .	11 " .			
11	Severe, 1 . .	3d. .	6 " .	" 23, 1901	1	"
	Severe, 1 . . .	3d. .	9 " .			
	Severe, 1 . . .	5th .	14 " .			
12	Moderate, 1. .	3d. .	12 " .	" 26, 1901	1	" Repetition of No. 14.
	Well marked, 1	2d. .	10 " .			
13	Moderate. 1. .	3d. .	15 " .	" 29, 1901	1	"
	Well marked, 1	2d. .	13 " .			
14	Severe, 1 . . .	2d. .	10 " . .	" 26, 1901	1	" Repetition
15	Severe, 1 . . .	2d. .	13 " . .	" 29, 1901	1	"
16	Severe, 1 . . .	2d .	16 " .	Dec. 2, 1901	2	"
	Fatal, 1.. . .	3d. .	12 " .			
17	Well marked, 1	3d. .	12 " . .	Nov.26, 1901	1	" Repetition
18	Well marked, 1	3d. .	15 " . .	" 29, 1901	1	"
19	Well marked, 1	3d. .	18 " . .	Dec. 2, 1901	2	"
20	Mild, 1	1st. .	4 " . .	" 17, 1901	14	" Repetition
21	Mild, 1	1st. .	11 " . .	" 24, 1901	7	"
22	Very mild. .	8th hr.	82 F., 22 days.	Jan.25, 1901	12	"

NOTE.—The subjects of Experiments Nos. 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, and 21 were proved to be susceptible by subsequent experimental infection. No. 22 declined a second test. In Experiments 20 and 21 the same set of insects and the same subject were used. Four of these insects infected this man six days later, on Dec. 30, 1900.

20. Case 8 of Dr. Guiteras' series, Amer. Med., Nov. 23, 1901.

21. See reprint, p. 8, on "Method of Stamping Out Yellow Fever, Etc.," by Dr. Charles Finlay, M.D., Conference of State and Provincial Boards of Health, New Haven, Conn., Oct. 20, 1902, and Medicine, Detroit, March, 1903, pp. 175, 179.

too late, after facts have been proved and recorded by others, to claim priority unless one can point to unmistakable evidence, in print, as proof that the facts have been demonstrated and duly reported. In our preliminary report[22] Dr. Finlay has been given due credit for priority in advancing and advocating the mosquito theory. Nothing more is due to him. He states[21] that he had "many years ago discovered that yellow fever was transmitted by mosquitoes," when in reality he simply assumes that to be the case, and the facts recorded by him do not sustain his claim.[23] He further states[21] that his own work had been singularly misrepresented before the American public, and that "among the facts and conjectures which are attributed to the recent investigators there is scarcely one which had not been asserted, demonstrated or suggested by me, as the result of my personal experiments and observations." One can only express admiration for this eminent scientist's persistent adherence to his theory, but the unbiased observer, after a perusal of his numerous writings, must agree with us that while he asserted and suggested everything that intelligent reasoning and profound knowledge could suggest, he failed to produce a single case of the disease. Our results have been confirmed by Guiteras with eight cases and three deaths;[24] Dr. Finlay's assumed results have never been confirmed by a single investigator. In our "Additional Note,"[25] we report among other instances one in which the bites of fourteen insects, applied four days after contamination, failed to infect, and the seven remaining alive failed also to infect eleven days after contamination, but on the seventeenth day following contamination the bites of four of the same insects produced an attack of yellow fever in "the same individual." During the whole of this time these insects had been kept

22. Phila. Med. Jour., Oct. 27, 1900.
23. Sternberg, Amer. Jour. Med. Sci., new series, vol. cii, 1891, p. 627, and Nuttall, Johns Hopkins Hosp. Reports, vol. viii, 1899-1900, p. 27.
24. Amer. Med., Nov. 23, 1901.
25. The Journal A. M. A., Feb. 16, 1901.

TABLE 3.—POSITIVE MOSQUITO INOCULATIONS OF THE ARMY COMMISSION.

No.	Character and Number of Patients Bitten.	Days of Disease.	Period in Mosquito.	Number of Mosquitoes Used.	Date of Application of Mosquitoes.	Incubation.	Character of Attack.
1	Severe, 1	2d	12 days	Room temp. 1	Aug. 27, 1900	3 days, 7 hours	Severe.
	Mild, 1	1st	6 days				
	Severe, 1	2d	4 days				
	Mild, 1	2d	2 days				
2	Fatal, 2	2d	2 to 16 days	Room temp. 4	Aug. 31, 1900	6 days	Moderate.
	Mild, 8	1st and 2d					
	Severe, 7	1st, 2d and 3d					
3	Fatal, 2	2d	2-15 days	Room temp. 5	Dec. 5, 1900	3 days, 10 hours	Pronounced.
	Severe, 1	2d	1-19 days				
	Mild, 1	3d	2-21 days				
4	Fatal, 1	3d	17 days	Room temp. 4	Dec. 8, 1900	5 days, 17 hours	Mild.
	Severe, 2	2d and 3d	18 and 22 days				
	Moderate, 1	3d	24 days				
5	Fatal, 1	2d	19 days	1 (Room temp.)	Dec. 9, 1900	3 days, 12 hours	Mild.
6	Fatal, 1	3d	20 days	Room temp. 4	Dec. 11, 1900	3 days, 20 hours	Moderate; relapse on 3d day.
	Severe, 2	2d and 3d	21 and 25 days.				
	Moderate, 1	3d	27 days				
7	Severe, 1	2d	1-24 days	82 deg. F. 15	Dec. 21, 1900	3 days, 23 hours	Pronounced.
	Well marked, 2	1st	10-5 and 12 days				
	Mild, 1	1st	4-8 days				
8	Mild, 1	1st	17 days	4 (82 deg. F.)	Dec. 30, 1900	3 days, 23 hours	Mild.
9	Well marked, 1	3d	39 days	3 (82 deg. F.)	Jan. 19, 1901	4 days	Moderate.
10.	Well marked, 1	3d	51 days	2 (82 deg. F.)	Jan. 31, 1901	3 days, 3 hours	Mild.
11	Well marked, 1	3d	57 days	2 (82 deg. F.)	Feb. 6, 1901	3 days, 6 hours	Mild.
12	Fatal, 1	2d	16 days	3 (82 deg. F.)	Feb. 7, 1901	2 days, 22 hours	Moderate.
13	Fatal, 1	3d	1-53 days	4 (Room temp.)	Sept. 16. 1901	3 days	Severe.
	Fatal, 1	2d	3-34 days				
14	Severe, 1	2d	18 days	8 (Room temp.)	Oct. 9, 1901	3 days, 8 hours	Mild.

in a heated room at an average temperature of 82 F. If, as is claimed by Finlay,[26] protection is conferred by one or two bites of "recently contaminated" (two to five days[19]) mosquitoes, this individual, who had received twenty-one bites from recently contaminated insects, should have been able to resist the bites of four of the same insects six and thirteen days later. Should one accept Finlay's extension of the incubation period to twenty-two days infection might then be erroneously attributed to the bites inflicted thirteen days before the onset of fever.

The Army Board worked on an entirely different hypothesis from Finlay, who believed that the infecting agent was retained on the biting parts of the mosquito, that infection was direct and no interval was necessary. The board worked on the hypothesis that the specific agent of yellow fever was probably one of that group of strict parasites that in nature necessarily pass through two distinct and alternating cycles of development, one within the body of a vertebrate, the other within a blood-sucking invertebrate host. Finlay believed that the bite of a single insect would confer a mild infection and multiple bites a severe one. In my own case, produced by the bite of a single insect, a fatal result was looked for during several days. Dr. Lazear was bitten by a single insect and died.[27] I became so firmly convinced that the severity of an attack depended on the susceptibility of an individual rather than on the number of bites sustained, that on Oct. 9, 1901, at Havana, I purposely applied to a non-immune eight mosquitoes (all I had) that had been contaminated eighteen days before. The attack that followed was a mild one, the temperature never reached 103 F., and dropped to normal on the evening of the fourth day.[27]

The original theory of Finlay has been variously modified by him from time to time to meet possible con-

26. Finlay: Edinburgh Med. Jour., Oct. 1894, p. 335.
27. The Etiology of Yellow Fever, Amer. Med., Feb. 22, 1902, Case 2.

tingencies. He now evidently inclines to the belief that development of the germ takes place in the salivary glands of the infected mosquito.[21]

A complete proof or disproof of his claim of positive results for his recorded inoculations could easily be obtained by means of a series of innocent experiments. It is interesting to note that at the very time when this distinguished authority is in position to confirm the results of his first inoculations, if they are susceptible of confirmation, he takes the ground that inoculation experiments on human beings in Cuba must be strictly forbidden.[21]

TABLE 4.—POSITIVE MOSQUITO INOCULATIONS OF DR. GUITERAS.

Number.	Character of Attack and Number of Patients Bitten.	Day of Disease.	Period in Mosquito.	No. of Mosquitoes Used	Date of Application of Mosquitoes.	Incubation.	Character of Attack.
1	Severe, 1	1st	26 days	1	Feb. 23, 1901	3 days, 10 hours	Pronounced
2	Severe, 1	3d	19 "	4	Aug. 8, 1901	4 " 5 "	Fatal
3	Severe, 1	3d	19 "	4	Aug. 8, 1901	3 " 3 "	Mild
*4	Severe, 1	3d	20 "	3	Aug. 9, 1901	5 " 3 "	Fatal
5	Severe, 1	3d	24 "	1	Aug. 13, 1901	3 " 19 "	Mild
6	Severe, 1	3d	24 "	2	Aug. 14, 1901	3 " 21 "	Fatal
7	Severe, 1	3d	24 "	3	Aug. 14 1901	5 " 21 "	Moder'ly severe
8	Severe, 1	3d	34 "	2	Aug. 22, 1901	3 " 0 "	Severe

*Case 4 had been bitten with negative result ten days previously by four mosquitoes five days after they had bitten a fatal case in the third day. of the disease. See Case 38 of Guiteras' table, Amer. Med., Nov. 23, 1901, p. 811.

It is unfortunate that Dr. Finlay in his numerous contributions to this subject very seldom gives exact references that would enable one to compare his more recent assertions with his former statements. In regard to his expressed opinion that the great merit of our work consists in our having accurately defined the danger line, beyond which there is a risk of producing severe or fatal experimental cases when non-immunes are inoculated with mosquitoes whose contamination is more than ten

days old,[21] I desire to record my belief, based on experimental observation and on the results obtained by Guiteras, that it is impossible to confer protection or to produce infection of any grade, with an insect that has been contaminated less than a week, and that inoculations with a mosquito contaminated for a shorter period will be absolutely devoid of result. The accompanying tables, compiled from our and Guiteras' cases, afford a substantial basis for the assertion. I deem it a duty to the service to which I belong, to my dead colleagues (Reed and Lazear) and to the profession, to state the facts plainly and clearly. I do not expect that this paper will remained unanswered; nevertheless, I shall decline to engage in controversy and leave the final determination to those who are competent to judge, after they have carefully perused the publications herein referred to.

The mosquito theory has been practically demonstrated by the army commission and Dr. Guiteras in two series comprising twenty-two cases in all. The non-communicability of the disease by means of fomites has been a matter of common observation in certain localities, and has also been demonstrated practically by our own experiments and those of Havard and his associates. Yellow fever, therefore, is not a filth disease, in the strict sense of the term, and filthy accumulations are dangerous in this connection only, when accompanied by sufficient moisture to breed mosquitoes under favorable conditions of temperature. Under such conditions standing water is a source of danger, within dwellings or without, and whether dirty or clean, unless it be kept covered or is changed every few days. Yellow fever is a preventable disease; quarantine, if effective, will exclude it, but if the quarantine be evaded and cases gain entrance to a city the disease can be absolutely controlled and finally exterminated by measures directed against the mosquito alone.

Carter[7] has well shown[8] that first cases are not dan-

gerous until after the lapse of a number of days, for, in his experience, persons in direct contact with them for a few days only never developed the disease after being placed in quarantine. This applies only to the first cases occurring in a town or neighborhood. He found that secondary cases did not appear until the expiration of a number of days varying from 11½ to 29, the average being eighteen. This interval represents the time necessary for the mosquito, after biting a patient, to become capable of infecting, plus the period of incubation in the person bitten.

The greatest obstacle to the control of this disease is the failure to recognize the first cases when they are not accompanied by black vomit. Some of these cases are so mild in character that no physician would dare to pronounce them yellow fever unless he knew the disease to be prevailing at the time. Susceptible persons visiting the house or living in adjoining houses would be bitten and develop the disease in near or distant localities several weeks later, forming secondary foci of infection. From these secondary foci tertiary foci would in time develop. The diagnosis of bilious remittent fever, dengue, etc., would serve to cover the earlier cases until the appearance of black vomit, by which time the infection would have become more or less widely disseminated. Such is the history of many outbreaks.

The number of mosquitoes in infested districts can always be appreciably diminished by giving attention to surface drainage and the removal of standing water, no matter how small the quantity or how pure the quality may be. Rain-gutters and conduits on buildings should be kept clear and free; cisterns, wells, flush-tanks and other permanent receptacles for water should be kept tightly covered or securely screened; privies and cesspools should be cared for; air vents to cisterns and wells protected by screens; and water should never be permitted to stand in any vessel or receptacle within a building longer than three or four days. Ditches and

open drains containing water should be frequently flushed; old cans, crockery, etc., should be removed from the vicinity of dwellings; pools or ponds should be stocked with fish and deepened at the margins or treated with petroleum. As the stegomyia is largely a house-dwelling and a house-breeding insect, these and other similar measures will surely diminish their number and add to the general comfort and protection. In places exposed to yellow fever infection all fever patients should be protected, night and day, from the bites of mosquitoes by the use of netting and wire screens. Strict quarantine measures, local and general, with the isolation of patients and extensive and thorough fumigation of the localities where they are found will insure suppression of the disease wherever it may appear.

In the suppression or control of yellow fever disinfection, apart from fumigation, is a waste of time and money. Natural yellow fever is transmitted by the mosquito, and always and only by the mosquito. The harmlessness of fomites has been fully demonstrated by our experiments in 1900 and 1901, in which three young Americans slept for twenty consecutive nights in a room garnished with articles soiled with black vomit, bloody fecal discharges and urine from fatal and other cases of yellow fever. Three and four large boxes were packed and unpacked with these articles night and morning by these non-immunes, who suffered no disturbance of health from these exposures. The room was twenty feet by fourteen feet, double walled, tightly ceiled, heated to above 90 F. and dark. Water was always present to keep the atmosphere moist. Two other non-immunes then occupied the room for twenty nights while additional articles of bedding and clothing were added. They slept in the garments and between the sheets that had covered cases of yellow fever, some of which were fatal. The result of this second exposure was *nil*. A third attempt was then made with two additional non-immunes equally without success. Not the slightest indisposition

followed close and intimate contact with this repulsive material in any case. Temperatures and pulse rates were recorded at regular and frequent intervals. Four of these seven non-immunes were subsequently infected by blood injections and by means of infected mosquitoes. A fifth resisted the mosquito once[28] and declined further attempts at inoculation.

Yellow fever has been eradicated from Havana, one of its endemic homes, by the institution of measures directed against the mosquito, after extreme cleanliness and energetic disinfection had proved a dismal failure. Several cases subsequently imported into that city have been handled with impunity by guarding the patients against the bites of those insects. A small outbreak in Santiago de las Vegas in 1901 was promptly suppressed by Colonel Gorgas by the use of means directed only against the mosquito. On those who decline to accept such evidence must rest the burden of proving that the disease is transmitted in some other way before their position can be sustained.

28. Case 22, Table 2.

THE STEGOMYIA AND FOMITES IN YELLOW FEVER.

STANFORD E. CHAILLE, M.D.
NEW ORLEANS.

As a student since 1851 of yellow fever, especially of its causation and prevention; and as a citizen of New Orleans, who has shared its blighted prosperity and its grief, greater, because of this disease, than has afflicted any other place in our country, I deem it my first duty to express most profound and grateful appreciation of those who have, since 1900, contributed facts of incalculable value for prevention and for commerce.

The notable experiments of Dr. Walter Reed and his assistants, Drs. James Carroll and A. Agramonte—added to and strengthened by the researches and labors of Drs. Carlos J. Finlay and John Guiteras, of Drs. W. C. Gorgas, John W. Ross, H. R. Carter and probably others—and rendered possible by the official encouragement and aid of Surgeon-General Sternberg, U. S. A., and of Governor-General Wood, M.D., have contributed to our knowledge of yellow fever facts that I hope and believe will prove of greater value than all the facts gained by all preceding students of the disease. The names cited deserve the earliest place on the roll of honor of the twentieth century, the century probably destined to become unprecedentedly illustrious for additions to the best of all knowledge, that which contributes to the health and welfare of mankind.

These investigators have contributed four sets of precious facts:

First—Fresh blood or its serum, taken from a case of yellow fever during the first days of the attack, will give the disease, if hypodermically injected into a non-immune. Hence the blood contains the poison, probably an ultra-microscopic germ, as I maintained in 1880, and this poison, when fresh, is infective without undergoing any change outside of the body.

Second.—The female stegomyia fasciata mosquito, serving as an intermediate host, can convey the poison to non-immunes. In twenty experimental cases (Reed's twelve and Guiteras' eight) the stegomyia became infected by sucking the blood during the first four days of the attack, but not later; and an interval of at least twelve days was required to infect a non-immune. However, it deserves note that no one has stated or proved that more numerous experiments might not have yielded some cases wherein the four days' time would have been prolonged and the twelve days' interval shortened. The facts thus far stated have not, I think, been denied by any one.

Third—Excluding all mosquitoes, fomites prepared and kept under all the known conditions, universally supposed to be the very best, for supplying the most virulent fomites, failed in December and January, though an artificial summer heat was maintained, to infect seven non-immunes extraordinarily exposed to said fomites. In a second trial the following September and October, 1901, under like favorable conditions, eight non-immunes also escaped infection. These negative results in two trials, involving fifteen non-immunes, have been confidently claimed to be conclusive of the impotency of fomites ever to infect; and this claim has been as confidently denied, because, as is said, of many facts explicable by fomites but inexplicable by the stegomyia.

Fourth—Disregarding filth and fomites, because two years of warfare against them had proved ineffectual, and limiting sanitary measures solely to excluding imported cases, to isolating every home case that did occur

and to preventing in every possible way the access of mosquitoes—these measures were followed in Havana by the total disappearance of all home cases from Sept. 28, 1901, to May, 1903.[1] Note in this connection that not less than eight cases have been imported during this time, but these cases have been isolated and carefully protected from mosquitoes, and that there have been in Havana an abundance of non-immunes. Now these four facts, introduction of imported cases, presence of many non-immunes, protection from mosquitoes and freedom from yellow fever in Havana, annually infected for at least 140 years, and monthly certainly for forty-five years and probably longer, were either cause and effect or the most extraordinary coincidences in the history of yellow fever. The more numerous such coincidences may become the stronger the proof of cause and effect. Several such coincidences have already been reported, viz., at Las Animas Hospital, Havana, 1899 and 1900, at Las Vegas, 12 miles from Havana, 1901, and at Havana, 1901, 1902 and to May, 1903.

Further, freedom from yellow fever, though stegomyiæ were present, would not be conclusive evidence against them; though present they might not be infected; and, if infected, very many would die prior to the twelve days requisite to become infective; and those that lived long enough to become infective might fail to gain access to non-immunes, and such failures would be the more probable the more numerous the immunes. Still further, the following reports are suggestive. It is now generally conceded that without anopheles there is no malaria. However, localities are reported from Italy, France and other countries where, although malaria once prevailed, it no longer occurs, and yet this failure to occur coexists with the annual importation of malarial

1. The warfare against mosquitoes in Havana was begun Feb. 27, 1901; preventive measures against filth and fomites were abandoned July 1. There were twelve deaths in January and February, but only six deaths March 1 to Sept. 28, 1901, and none since; with the sole exception that of seven *imported* cases of yellow fever in 1902, two died.

cases and the continued presence of many anopheles. And a locality is reported in England where anopheles refused to suck man's blood.

The case of fomites versus the stegomyia is of the utmost importance for prevention and commerce and now deserves consideration. The only advocates of fomites known to me who have published their views are two distinguished sanitary officers, having much knowledge of and experience in yellow fever, Dr. John H. Purnell of Mississippi and Dr. Edmond Souchon, president of the Louisiana State Board of Health. The arguments of one or the other, to prove that the stegomyia is not the sole cause, but that fomites are also a cause, will be summarized also under four heads.

First—It is claimed that the freedom of Havana from yellow fever in 1901 and 1902 was not due to the warfare on mosquitoes, but to the rigid exclusion of imported cases and to the dying out of the disease. In support of this claim there is some evidence that other Cuban cities escaped yellow fever without effective warfare against mosquitoes. But, even if this evidence were indisputable, there are good reasons for believing that imported cases were excluded, that any cases that did occur were isolated and access of mosquitoes to them was prevented, and that these cities had very few non-immune inhabitants. For such reasons these cities may have escaped yellow fever consistently with the stegomyia theory, although these cities may have failed to wage effective warfare against mosquitoes.

The tendency of yellow fever, as of other infectious diseases, to die out, in spite of the coexistence of some cases and of many non-immunes, has been manifested many times even in the distant past, when imported cases were not excluded and when there was no warfare against filth, fomites or mosquitoes. A cause of this tendency of yellow fever may be a scanty reproduction in some years of the stegomyiæ, as occurs with other insects. In spite of this tendency, it is certain that this has failed for 140

years to manifest itself in Havana to an extent at all comparable with the remarkable cessation of the disease since warfare against mosquitoes was begun. Hence, the extraordinary escape of Havana, the worst and the longest infected city in the world, inclines the probabilities strongly in favor of the claim that its rescue from yellow fever is due to warfare against the stegomyia.

Second—The frequent return or recrudescence of yellow fever six, twelve or more months after a cold winter and without the reintroduction of the infection, is incompatible with our present knowledge of the duration of life of the stegomyia. The greatest duration yet reported was found by Dr. John Guiteras in 1902. Of eleven infected stegomyiæ two died by the tenth day, five were dead by the sixty-fifth day, and of the remaining six the last one lived to its one hundred and fifty-fourth day. It deserves special notice that 18 per cent. died before the twelfth day, i. e., too soon to have become infective, and that this mortality occurred in captives well protected from the many enemies mosquitoes are exposed to in the open. Dr. H. A. Veazie has reported that of one hundred New Orleans mosquitoes one-half died in the first week and only two were alive on the thirtieth day. Manifestly a mosquito's life will vary with its protection from enemies, with its food, temperature, light and other variable conditions. How long an infected stegomyia may possibly live under the influence of hibernation and other exceptional conditions is yet to be determined. However, Dr. John Guiteras[2] has reported that of thirty-three female stegomyiæ kept in an icebox at 40 to 50 F., and without food or water, three lived to the eighty-seventh day and were then destroyed by ants.

My consideration of recrudescence will be limited to criticisms on one example cited oftenest, cited recently by Dr. Purnell and by Dr. Souchon, cited also by Dr.

2. Revista de Medicina Tropica of Havana, April, 1903.

Sternberg and by myself in 1880, when, however, every one believed in fomites and no one in the stegomyia. Hence, my reinvestigation of the supposed recrudescence in Memphis in 1879, after its epidemic of 1878, the most fatal one, proportionately to population, ever in the United States.

The best authority for this recrudescence was Dr. G. B. Thornton, the thoroughly trustworthy president of the Memphis Board of Health. He reported[3] that the first case was attacked about the 5th, was reported on the 8th and died the 9th of July; that, although in all preceding epidemics the first case was known to have been imported from New Orleans, yet in 1879 no such case was discovered; and further, that yellow fever was not then "prevailing" in any part of the United States. From these premises Dr. Thornton and most of the medical profession concluded that the disease was due to "local origin," which, in this case, meant to houses and their contents infected in 1878, i. e., to the recrudescence of the dormant vitality of infected fomites. A conclusion in full accord with the professional opinion of 1879.

To these facts and to this conclusion I now add, for the first time, I believe, in this connection, the three following facts: Dr. W. L. Coleman,[4] an inspector of the National Board of Health, maintained that the disease was certainly due to a fresh importation of fomites, viz., to two box-cars of bananas from the West Indies, via Havana and New Orleans, that arrived in Memphis prior to July 4, 1879. Further, Dr. Thornton's final conclusion was that "the disease may have been imported. If such was the case, the epidemic was, in my opinion, due to two causes, importation and local origin." Still further and more important, while it was correctly stated that yellow fever was not "prevailing" anywhere

3. In Vols. v and vi, Transactions of the American Public Health Association and in recent letters to me.

4. See "Yellow Fever and Dengue," by W. L. Coleman, M.D., pp. 100-2.

in the United States, none the less it did exist in New Orleans, as proved by the annual report of the Louisiana State Board of Health for 1879, wherein is recorded one imported case, March 26, one home case on the 16th and another on the 17th of June. Evidently, then, there was time sufficient for stegomyiæ to be transported, by the open communications then existing, from New Orleans to Memphis to infect the first case reported in Memphis. These facts prove that this alleged recrudescence might have been due to stegomyiæ and not to fomites. Conjoin the indisputable fact that the first cases reported in New Orleans have often not been the first that occurred and the case of the stegomyia becomes still stronger. It is doubtful whether, if all the facts could be known, other instances of recrudescence by fomites would not become as questionable as the most often cited of all instances, Memphis in 1879.

Third—In behalf of fomites it has been urged that non-immunes in camps of refuge, infested by mosquitoes and near infected cities, have been kept free from infection by the destruction or disinfection of fomites. This conclusion is very far from proven. Such cases of the disease as were imported were usually promptly isolated; quite possibly there may have been swarms of mosquitoes, but no stegomyiæ, for these seem to prefer houses and to "shun rural districts and open fields"; and there may have been stegomyiæ but none infected. Further, New Orleans could furnish many instances of adjacent places in the country, and of hospitals, asylums, jails and convents within the city, into which places cases of yellow fever were introduced but failed to originate any cases though fomites were neglected. Such exemptions occurred frequently in the distant past, when no efforts were made either to isolate the disease or to disinfect fomites. It seems to me that all such exemptions are more explicable by the absence of infected stegomyiæ than by the fomites theory.

Three instances, often cited, of exemption from yellow

fever will illustrate the claims of advocates of fomites. In 1878 many hundreds of refugees fled from Memphis to Camp Joe Williams, four and a half miles distant. Fomites were destroyed or disinfected, and although there were imported into this camp 186 cases of yellow fever, of whom 58 died, yet no cases originated in this camp. However, Colonel Cameron,[5] commander of the camp, reported the following facts: "Four grave diggers and one carpenter, on constant duty at the hospital carrying the sick, washing the bodies, sleeping on infected bedding in tents, wearing clothing stripped from the bodies of deceased, remained on said duties seven weeks with perfect impunity from attack." "None had previously had fever." Now if many refugees owed their escape from yellow fever to destruction of fomites, what protected these five non-immunes from fomites not disinfected?

The epidemic of 1878 in Memphis, with not one-sixth the population of New Orleans, caused over 5,000 deaths, while there were less than 5,000 in New Orleans. However, in 1879 Memphis had "1,532 cases and 485 deaths" by yellow fever, while New Orleans had only 48 cases and 19 deaths, and 34 of these cases and 12 deaths were restricted to a limited section of the city. Now, this comparative exemption of New Orleans was claimed to be due to the vigorous co-operation of the state and national boards of health and the Sanitary Association in the repeated cleansing and renovation of infected places, in thorough disinfection and in the sulphur fumigation of houses.

Further, in 1882 there were only four reported deaths in New Orleans, and this comparative exemption was claimed to be due to thorough cleansing and disinfection of the section surrounding the focus of infection "for at least four blocks each way around the focus." Now, it is notable that in the last two instances sulphur

5. Transactions of American Public Health Association, p 155, vol. v.

fumigations were vigorously applied—the very remedy specially recommended to destroy stegomyiæ.

Fourth—Reported cases of two kinds have been urged as explicable by fomites, but inexplicable by stegomyiæ. Some cases of yellow fever have been reported where the possible infection of stegomyiæ and the transfer of the infection to the person attacked gave intervals of only 8, 9 and 10 days instead of the least interval of 12 days found in the twenty experimental cases. More experimental cases might have proved the possibility of a less interval than 12 days; but a more serious criticism is that the briefer intervals were based on supposed knowledge of what was the very first case, very often too doubtful to justify a final conclusion.

Numerous other cases are reported to have followed, within the usual period of incubation, exposure to fomites under conditions such that the presence of any infected mosquitoes was highly improbable if not impossible. In some of these cases the disease was limited to the few non-immunes exposed to the fomites, while other non-immunes in the very same house were not attacked. In such cases it is a notable and suspicious fact that even the fomites from those attacked failed to infect the other non-immunes in the same house.

All of these reported cases, as well as some other issues, are amenable to the two following criticisms: Every reporter seeking a cause is not only apt to find it in whatever may be universally admitted to be a cause, though not one, but is also apt to overlook a universally unsuspected cause, though this be the true one. There have been many reported cases of disease explained satisfactorily to reporters and their readers by witches, the moon, comets, bile, urea and other erroneous causes. Without anopheles malaria does not occur, yet the very word falsely assigns bad air as the cause, and this disease has also been often attributed not only to bad water, but also to watermelons, and these causes have been maintained by evidence satisfactory to reporters and their readers.

Many cases of yellow fever in adults, many more in children and negroes, can not be diagnosed by the best experts, and much more numerous must be the cases undiagnosable by the great majority of physicians who are not experts. Over fifty years' study of yellow fever, eight of these years living day and night in the hospitals of New Orleans, when the disease annually prevailed, and six more of these years employed as a United States sanitary officer specially for yellow fever, and given unusual opportunities for investigation, would justify me in swearing in court that the first cases officially reported in a civil population are very often and, as I believe, usually not the first cases that occurred. This is due not merely to diagnostic deficiencies, but also to two other causes, viz., lack of opportunity among the numerous poor unvisited by a physician, and the very frequent neglect of physicians to report their first cases, even when diagnosed. This neglect is due to their unwillingness to become, in public estimation, odious alarmists and also to subject their patients and the families of their patients to disagreeable notoriety and often to legal restraint by sanitary officers. Practical sanitarians are forced to recognize that the more the financial welfare of the public is threatened by the invasion of a communicable disease and the greater the legal restraint imposed on the public and on the sick and their families, the fewer will be the cases reported. For reasons now stated conclusions based on supposed knowledge of first cases are often untrustworthy.

Three more sets of facts pertinent to my subject deserve brief attention. Dr. H. R. Carter,[6] the author of the best summary known to me of arguments in favor of the stegomyia, urges this very weighty generalization. He claims that every disease having an intermediate host is propagated solely by its special host and never by fomites. Though convinced that present knowledge

6. Bulletin No. 10, Yellow Fever Institute of U. S. Bureau of Public Health and Marine-Hospital Service.

favors this generalization, I doubt whether we yet know enough to justify a final conclusion. In any case, claims for the infectivity of fomites would be more explicable should knowledge enlighten our ignorance in the following particulars.

Of these diseases malaria and the Texas cattle fever spread by a special tick, are best known in our country and most resemble yellow fever. It deserves note that the tick fever, besides other resemblances to yellow fever, has these two striking ones, the young have usually very mild attacks and an attack confers immunity. Now, the infecting germ persists in malaria often for months and years and in cattle fever probably for life; and this long persistence of the germ in healthy immune cattle caused until recently the erroneous belief[7] that infected ticks infected their own eggs. However, my present point is that while the blood in malaria and in cattle fever remains infective long after the first attack has ended, yet the twenty experimental cases indicate that the blood of a yellow fever patient is infective during only the first four days of the attack. The lack of resemblance thus indicated justifies the suspicion that a closer resemblance may yet be proved. A ray of light may yet be found in Finlay's belief that in malaria man is the permanent and anopheles the temporary host, while in yellow fever man is the temporary and the stegomyia the permanent host.

Further, Dr. Patrick Manson, one of the best authorities on malaria, gives good reason for his beliefs, that the malarial germ may have other host than man, and that this germ may be deposited in the soil, inhaled in dust and thus cause the disease. And many good observers still maintain that drinking water may become infectious by the dying therein of infected mosquitoes, as is still taught by some to be the case in filariasis. Still

7. According to Dr. D. E. Salmon, Chief of U. S. Bureau of Animal Industry.

further, Dr. Carlos J. Finlay believes that the fresh blood of yellow fever on the proboscis of a stegomyia can infect a non-immune within the usual period of incubation, while the germs within the stegomyia would require twelve or more days to become infective. I have now indicated five particulars, as to which future knowledge may throw light on the causes that have led to belief in fomites.

The history of the remarkable change of view in New Orleans as to belief in fomites is instructive. A great majority of its physicians, myself included, firmly believed, until at least 1867, that yellow fever originated in this city, as was then universally claimed as to other habitually infected cities, and all of this great majority unhesitatingly testified that they had never observed a single case either of direct contagion or of infection by fomites. As steamboats and railroads multiplied and many adjacent places on the routes of travel became infected *after* outbreaks of the disease in New Orleans, the conviction gradually gained ground that yellow fever did follow the routes of travel and was an infectious disease. Then followed numerous reports that some places on these routes had not had any case of yellow fever introduced therein, certainly the usual precursor of an outbreak, but that fomites had been introduced. Our imaginations failed to invent any explanation for these credited reports except infection by fomites. However, this concession to fomites was made very grudgingly, because New Orleans physicians could not ignore their innumerable experiences of the failure of fomites to infect. Hence the hypothesis invented to explain these credited reports was that, while fomites were usually innocous yet that under the rare influence of some unknown mysterious conditions fomites did become infectious.

For such reasons I, as soon as convinced that mosquitoes did follow the routes of travel and that the stegomyia was a conveyer of yellow fever, embraced the

stegomyia as a welcome substitute for those unknown mysterious conditions about which I have wasted, as I now believe, many weary hours of study; and I earnestly hope that the future may permit my brain to rest in peace with the comprehensible stegomyia—unvexed by the incomprehensible, mysterious hoodoo, fomites. I, like others, have advocated fomites, always, however, citing my authority. For my part, I have never secured satisfactory proof of a single case either of direct contagion or of infection by fomites. I have investigated reported cases of infection by fomites, and I have known several cases, supported by very strong evidence, which, after laborious investigation, were conclusively traced to a usual cause, viz., to infected houses, wherein were women of easy virtue. These had received night-visits, carefully concealed and denied by men who, living in uninfected country places, had contracted the disease in said infected houses, and not by fomites in their uninfected country places.

Finally, it deserves consideration in behalf of sanitary officers who hesitate to abandon the disinfection of fomites, that more places than New Orleans have suffered by numerous advocates of innumerable "sure cures" and sure preventives for yellow fever. Notable among these advocates was Gen. Benjamin Butler, U. S. A., in 1862. He, whose memory is still odious in New Orleans, issued in 1863 a pompous farewell address in which he proclaimed: "I have demonstrated that the pestilence can be kept from your borders." His sure preventive was cleanliness, enforced by sanitary regulations, executed by an efficient sanitary police. However, conjoined with this sure preventive the U. S. Navy effectually blockaded New Orleans. General Butler's sure preventive was heartily indorsed by many medical officers of the Army, by northern physicians generally, by public and medical press and by many eminent sanitarians. Among these was Dr. Elisha Harris of New York, who had held several of the highest positions as a

sanitary officer and became a president of the American Public Health Association. His advocacy was so conspicuous and enthusiastic that I replied to General Butler's claim[8] and closed with this question, which has never yet been answered: If New Orleans was saved from yellow fever by General Butler in 1862, when there were two reported deaths, who saved New Orleans in 1861, when there was not one reported case or death, and when blockaded New Orleans was occupied by General Lovell and his very many non-immune Confederate soldiers, who enforced no preventives of any kind?

CONCLUSIONS.

My reasons have now been indicated for the five following conclusions: The probabilities now are that the stegomyia, the only cause yet proven, is the sole cause for the dissemination of yellow fever; but that time is required to convert these probabilities into certainty. Boards of health, should they neglect any practicable measure of warfare against the stegomyia, would deserve the severest punishment of the worst criminals. Southern boards of health are excusable for over-precaution against yellow fever. States adjacent to Louisiana have power, by sanitary restrictions, to strangle the commerce of New Orleans and Louisiana should not abandon the disinfection of fomites until approved by said states. Boards of health are representatives of the people and are justified in continuing to disinfect fomites as long as public opinion may favor this measure and until the probability of its inutility is converted into certainty.

To my many friends who urge that Mrs. Stegomyia has inherited all the claims of her father, Mr. Fomites, who is dead and ought to be buried, I tender the advice given at a numerously attended funeral by the undertaker: "Gentlemen, gentlemen, please don't crowd the mourners"—mourners over the remains of Mr. Fomites!

---

8. In the New York Sanitarian, August, 1882.

DISCUSSION

ON PAPERS OF DRS. CARROLL AND CHAILLE.

DR. EDMOND SOUCHON, New Orleans—It is impossible not to be deeply impressed by the reading of the experiments made by Reed and his associates in Havana. The simple, direct, positive, American-like manner in which these experiments were conducted can not but fill our minds with noble pride, as scientists, as men and as Americans. As such I would most probably have joined also the army of followers that have been conquered by the immortal Reed, but as the responsible officer of the Louisiana State Board of Health, the duty of which Board is to guard the valley of the Mississippi against the introduction of contagious diseases, I am compelled to carefully examine into every point, and to submit all the statements, past, present and future, to the crucial test of the Pasteur law, in accordance to which no facts should be accepted as thoroughly proven until all possible means to disprove them have failed. It is because Pasteur applied this law to his own work that not one of Pasteur's statements has ever been refuted.

At the outset, kindly note the words "responsible officer." The sense of responsibility for the awful consequences of possible errors changes the whole aspect of questions. The irresponsible ones are always quick to urge on. When they meet with reverses they naively say that they never would have thought that possible, and that is the end of it.

But when responsible officers have to face the introduction of contagious diseases, the press, the people, the authorities, all unanimously shout that they should have known better, should have been more prudent and more slow in applying new theories; and general condemnation is forever attached to their fair names. But a few years ago all these tragic events were enacted in this very city.

Then, there comes in also the overpowering consciousness of the dire calamities, the sufferings, the anxieties and the sorrows of sickness and the deprivations and ruins or quarantines. All this is properly calculated to cause responsible officers to be doubly careful before making any changes in quarantine regulations which may be followed by pestilence and quarantines.

The factor of responsibility is well illustrated by the following anecdote: A very high health official once had presented to him for signature a telegram relating to a very important quarantine matter. The chief scanned the prepared telegram most carefully, then said to his assistant that since he had charge of that department to sign the telegram himself. The assistant retired and it took him two hours to find the wording of a telegram which he would sign and be responsible for the conse-

quences. On going over the records of all the quarantine stations, state and federal, to which he could have access to and also over the scanty literature on the subject, the writer of the present was confronted with a number of cases of yellow fever where it was impossible for him and his responsible colleagues to see the intermediation of the mosquitoes, bearing in mind the fact that the mosquito does not live more than five days without water in the summer months.

We have collected 27 such cases in the meager literature of the day. Some are land cases and some are shore cases. However much I would like to read some of them to you, the time allotted me will not permit.

If such cases have been observed there have surely been many which have not been recorded. If such cases have presented themselves in the past, similar cases may again present themselves, and how can we safely ignore them in our quarantine regulations until they have been explained satisfactorily to the responsible officers.

To say that these cases are errors of diagnosis is begging the question. Reasonable men will admit that some of the cases may have been diagnosed wrongly, but reasonable men will also admit that it is unreasonable to say that all the cases were mistaken cases.

The first and foremost perplexity of the responsible officers was the question of the treatment of fomites. From the known experiments made by Reed and Carroll, Guiteras and Ross, it is contended that the disinfection of such is unnecessary. To these experiments the responsible officers oppose the fact that they were made at a time of the year when yellow fever is not at its worst, except perhaps the early experiments of Ross.

Then we must consider that 30 per cent. of the non-immunes bitten by infected mosquitoes did not develop yellow fever. It is, therefore, unreasonable to expect that all experiments made with fomites should develop yellow fever. Beside, these experiments with fomites were made by men; men of recognized ability it is true, but human all the same, and they may have overlooked some factor.

We hold that the cases reported of transmission of yellow fever without the intermediation of the mosquito are truly experiments made by Nature herself, and she surely has overlooked no factor.

A noted sanitarian mentions the fact that a large amount of fomites went from Cuba to New York and to Spain after the Cuban War and that no yellow fever developed in either country. But these countries are places where yellow fever develops with difficulty. If that baggage had come to New Orleans, Galveston or Mobile and no harm followed, that would mean something, but as it is it is of little value.

The responsible officers do not contend that it is fomites that transmit yellow fever. They modestly say that they do not know; that it may be some means of which no one thinks now until some day someone comes up against it as they did for the mosquito.

The evolution of the mosquito theory and demonstration has required nearly a quarter of a century and it may take also a long time before it is demonstrated that there is another means of transmission, as the facts related above strongly point.

To those who say that the mosquito was present outside of the baggage or fomites but was not observed because no one at the time thought of them or looked for them, the responsible officer replies that at present the mosquito partisans see mosquitoes everywhere, even where they are not.

We are told that the mosquito is the host of the yellow fever germ and that it is transmitted through the saliva of the mosquito; but this has never been proven since that germ has never been seen by anybody.

To the explanation that the mosquito being the host of the yellow fever germ it can only be transmitted from man to mosquitoes and from mosquitoes to man, the responsible officers reply that that would be true if it were demonstrated that the germ of yellow fever is an animal parasite, but this is yet to be done. No one knows if it is an animal or a vegetable germ. If a vegetable germ it can be transmitted in several ways. This is the law of Nature.

A noted sanitarian of the United States Public Health Service states that of such diseases as have been fully investigated, all that are conveyed from living hosts are carried by animal parasites. How about the plague whose germ is a vegetable organism and which is transmitted by animals it is true, but also by the air and by the food?

A good deal of stress is laid on destroying mosquitoes on board of all vessels, non-infected vessels as well as the infected ones. The truth is that the mosquitoes on non-infected fruit vessels are a very small factor in the transmission of yellow fever because the proportion of the stegomyia is only 2.5 per cent. That this small number is unimportant is borne out by the fact that the absence of yellow fever in the Las Animas Hospital before the mosquito era was explained later by Havard and Guiteras by the small number of the stegomyia found when the mosquitoes of the hospital were destroyed. Beside, the mosquitoes on board of non-infected fruit vessels are not infected. If they were there would have been more than one case of yellow fever not traceable to the shore in fifteen years on more than 1,000 fruit vessels. And there would have been yellow fever in New Orleans from 1886 to 1896, during which time 50,000 laborers were exposed to

mosquitoes while unloading fruit vessels before the fumigation of the holds.

The fact that a number of vessels without yellow fever at the port of departure or en route or on arrival at the home port have developed yellow fever after having been disinfected, points out also that there must be another means of transmission of yellow fever than the mosquito. Some of these vessels were many days out before arriving at the home port. If there were any infected mosquitoes on board they must have been well concealed that they did not bite anyone. Then how could they have lived without water, since they do not breed in bilge water or in sea water. There are many other contradictions, inconsistencies to cause perplexities, but we have no time here to dwell on them. In conclusion we will say that for the present and until further progress the responsible officers are unwilling to make any change in the quarantine regulations against yellow fever. They consider the question still under study and requiring more time and demonstration. Many believe, and the writer among them, that the demonstration of the unity of cause of transmission will most likely come, but it has not as yet arrived. Too many new ideas have been adopted, to be later exploded, not to cause responsible officers to move slowly in such momentous matters. Public opinion will require convincing proofs to stand by its quarantine officials when important changes will be proposed and specially put into practice.

The International Sanitary Conference held in Washington last December recognized that there were still some doubtful points regarding the transmission of yellow fever and that quarantine regulations should not be changed until further demonstrations.

Even if it should be demonstrated to the satisfaction of the responsible officers that the mosquito is the sole transmitter of yellow fever and that fomites truly do not convey it, the only difference it will make in the quarantine regulations is that the baggage, bedding, etc., shall not be subjected to disinfection and that the forecastle of vessels shall not be washed with bichlorid. The vessel shall have to be disinfected to destroy what mosquitoes are on board, and the crew and passengers shall have to be detained to make sure that no one is in the period of incubation. It is but little trouble to disinfect the baggage and bedding of a vessel compared to the possible results of the dispensation of it before more positive evidence shows that it is safe to do so.

Responsible officers are unwilling to run the risk of an epidemic with its consequent quarantine to prove that there is no other means of transmission of yellow fever except the mosquito. The needed further demonstrations will have to come some other way.

Dr. Quitman Kohnke, New Orleans—New Orleans is the most important city of the South, and the most important in this country in relation to yellow fever infection. Whenever yellow fever occurs in the South, New Orleans is accused of being the first city infected and the city from which it spreads.

We of New Orleans believe that yellow fever is always imported, and that, if recrudescence is at all possible, it occurs but seldom; we are, therefore, intensely interested in quarantine against yellow fever, though deprived by law of the power of quarantine, this authority residing in the State Board of Health of Louisiana.

I want to say here, to remove an erroneous impression, that no antagonism exists between the health authorities of the city and those of the state, save only that antagonism which may properly exist between two persons, differing in their views, but both tending in the same direction and both seeking the essential truth and the maximum protection which its application to preventive measures will insure to our people.

I may say, for instance, that I agree with Dr. Souchon in his determination to disinfect, for a while longer, for fomites for the sake of public opinion, the perfection of safety and the avoidance of injurious criticism (as Dr. Chaillé suggests); and I hope Dr. Souchon will agree with me in believing that the disinfection for mosquitoes should also be above criticism, and should be as thorough as it can possibly be made.

The recorded incidents of our quarantine history give us strong evidence corroborative of the mosquito transmission of the disease. Quarantine against yellow fever became practically effective only after the adoption in 1874 of Dr. Perry's method of sulphur fumigation of the holds of vessels, arriving from yellow fever ports. Mosquitoes were destroyed by this process, and the efficiency of the method was strikingly emphasized by the omission (by Dr. Perry's successor) to sulphur fumigate a vessel, on which, in consequence of this omission, yellow fever developed while the vessel was at wharf in this city; the vessels treated by Dr. Perry's method having escaped this experience.

I may express my views on this subject as the health officer of New Orleans in language previously employed for a similar purpose and say that, omitting from consideration the proper detention of inoculated persons, I believe firmly that all quarantines against yellow fever in the past, in so far as they have been successful in preventing the introduction of the disease, have been so because they prevented the entrance of infected mosquitoes. Whether this has been accomplished by a detention for a period longer than the life of the mosquito, or by the destruction of the insect in some other way, the achiev-

ing result has always been the excluding of infected mosquitoes.

The vagaries of the quarantine service, fluctuating from one extreme to another, actuated now by an exaggerated fear of infection, and again by powerful influences directed, perhaps unwittingly, to the interests of commercial circles, may well account for some of the phenomena of yellow fever visitations improperly attributed to other causes.

The recorded observations of men who knew nothing about the mosquito transmission of yellow fever form a chain of circumstantial evidence leading to conviction.

We have in New Orleans two abominations, of which we hope soon to be rid—the cistern and the water closet, both situated in the rear portion of our resident premises.

It has been observed that yellow fever travels from house to house more frequently over the back fence than to the side neighbors, and the close relationship between rear premises has for years been a matter of puzzling speculation. It is easily explained now by the mosquito theory. Cisterns are breeders of mosquitoes (stegomyia), and to the cisterns mosquitoes naturally go to obtain water. Some of these mosquitoes being infected it is easily supposable that they will fly from the cisterns or closets to the nearest neighbor, whose house may front on a street considerably distant from the previously infected house. It has also been observed in the past that the period of time intervening between house-to-house infection was in many instances about two weeks; this corresponds, also, to the mosquito transmissibility of yellow fever; and many of the recorded facts, other than these, and puzzling at the time of their observation, are made clear by the application of the mosquito theory to the otherwise unexplainable incidents.

The accumulated records of the past, viewed in the light of present knowledge, seem like a deliberate gathering of evidence against the mosquito; even before the thought of an indictment was entertained by those who laboriously and conscientiously (though not understandingly) piled up the mass of corroborating evidence, supporting the charge against the female stegomyia of transmitting yellow fever from one victim to another.

Dr. Felix Formento, New Orleans—Two years ago, at a meeting of the Public Health Association, which met at Buffalo, N. Y., a discussion on this subject took place in which Dr. Reed partook, a man who no one admired more than I did. The real subject for discussion was the communicability of the disease and the views that I then took are those that I take at the present time. It was then stated that communication occurred only through the bite of the mosquito. I think that the admirable work and the results of the work of the

American physicians in Havana have demonstrated that this disease is communicated by the bite of the mosquito. It has not been absolutely and positively demonstrated that yellow fever can not be communicated to man except through the mosquito; there have been cases in which the prevalence of the disease can not be explained by the mosquito theory.

A few years ago a trunk belonging to an officer from Havana was sent to Madrid. Several months later the trunk was opened, being several months after the death of that officer from yellow fever. It was soon after noted that members of that household, in which the trunk was opened, were taken down with yellow fever. There are other cases reported which seem to prove that there are other means of communication of this disease.

I think we should be more careful about laying aside such evidence. I have studied this disease since 1861, and there are many points regarding this disease that still are very obscure. During the past 12 years there have been no cases of yellow fever in New Orleans, where everything has been done against the mosquito. Cases have occurred at Vera Cruz and at Havana, but none have occurred here.

Formerly there was great opposition to quarantine being established and once it was asked that it be abolished; believing that the mosquito was the ordinary means of communication of this disease it was decided not to take such a decided measure as abolishing quarantine. I believe that we should be a little conservative in this direction.

Dr. C. W. Stiles, Washington, D. C.—I do not think there is anything strange about the conservatism that is found in New Orleans, as expressed by Dr. Formento; I should be surprised if it was not so. It is evident that the spirit of fairness that has been exhibited by members of the Association in New Orleans shows that the slight differences which they may have had is really ephemeral in nature.

There are certain biologic facts that should be considered in yellow fever. We all know that the cause must belong to one of three different groups, as follows: 1, bacterium; 2, flagella; 3, sporozoa. The entire nature of the disease, together with the short period of incubation, shows that the organism is reproduced by a non-sexual method and there are only three groups of organisms which reproduce in this way. The bacterium seems to be excluded because of the short period of incubation in the mosquito. Regarding the flagella nothing is known regarding its sexual development, but if there is any form of flagella that is reproduced by the sexual method, and if it is a cause of yellow fever, we must then admit that it is necessarily a wound disease. But we do not know any flagella in existence which can transmit the disease in this way. The only organism which corresponds to what we expect the

parasite should be, then, is the sporozoa. This organism is non-sexual. In every case the organism must pass through a definite and regular rotation.

Either yellow fever is caused by the sporozoa, or else it is caused by one of the other two mentioned; if the latter exist it must then be a coincidence. Then this period of incubation of 13 days means nothing at all. I prophesy that the disease will be shown to be a sporozoa disease and all differences of opinion will die out entirely so soon as this is shown to be the case.

DR. JAMES CARROLL, U. S. A.—I did not expect for a moment that my ideas would meet as much sympathy as is shown. We certainly appreciate the full responsibility that rests on the gentlemen who conduct the hygienic and sanitary affairs necessary to protect New Orleans from yellow fever. I wish to again make the statement that there is no other source of infection possible that has been proven. We have proven that this disease is transmitted by the mosquito. We have also proven that the disease is *not* transmitted by fomites.

When you consider the conditions under which the experiments were conducted at Havana one must be much impressed with the accuracy of them. Three non-immunes entered a building in which there were several articles of bedding, etc., which had been infected with the discharges of blood and fecal matter, urine, etc. They remained there during the night. In the morning they removed the articles from the ward, shook them and then repacked them in boxes. This routine was carried out every day for 20 days, when they came out in perfect health. Following them two other non-immunes entered. They slept there for 20 days and nights. They slept between sheets which had been used by fatal cases of yellow fever. In fact, everything imaginable was done to infect those men, and they would have become infected if such thing had been possible by such means. They, too, came out in perfect health. Other sets of non-immunes were tried. Even blood was drawn from infected patients and towels were soiled and these men slept with their heads between these bloody towels. They, too, came out in perfect health. Gentlemen, I believe positively that fomites are perfectly harmless. The material was kept there for several months and then removed without any precautions, sent to the laundry, and no one who handled them contracted yellow fever. Of 7 individuals experimented on, four subsequently contracted the disease from the bite of the mosquito and the subcutaneous injection of infected blood. There was no question as to the susceptibility of those infected. All were Americans. These experiments were reported in 1900, in August, by Drs. Guiteras and Ross, and another physician of Havana, and all with the same results.

After such experiments I contend that the burden of proof rests with the other side. On those who decline to accept this practical demonstration rests the burden of proof, and they must prove that the disease is transmitted in some other way. If it can be proven that the disease is spread by fomites we are willing to yield. I do not believe there is any doubt that our results will be confirmed, as they have already been so far by certain investigators. It has been found that those who suffer from the disease can be handled with impunity by nurses without any extension of the disease. Personally I believe that if there was no quarantine and no disinfection the disease could be excluded or prevented from spreading by proper precautions. Of course, that would result in placing everyone beneath a mosquito bar and protecting them by wire screens. Then there would be no possibility of spreading the disease. There are no cases of yellow fever now in Havana except imported ones. In regard to Dr. Souchon's statement with reference to the vessels arriving at the port of New Orleans after trips from South America and with the fever developing among the passengers five or six days after arriving at quarantine, they probably were infected after their arrival at the quarantine stations. There is an immense amount of literature on this subject.

ARTHRITIS DEFORMANS.

THE REPORT OF A SERIES OF ONE HUNDRED AND TEN CASES FROM THE JOHNS HOPKINS HOSPITAL.

(FROM THE CLINIC OF PROFESSOR OSLER.)

THOMAS McCRAE, M.D., M.R.C.P. (Lond.)
THE JOHNS HOPKINS HOSPITAL.
BALTIMORE.

There is no intention in this paper to make any attempt to discuss the many debatable points relative to arthritis deformans. There is, perhaps, no common disease in the calendar about which we have as little exact knowledge. Much misapprehension has been caused by the use of names which suggest that this disease has some association with rheumatism. Beginning with the very name to be given to it, we have great diversity of opinion, which continues through the etiology and pathology of the disease. Dr. Bradford[1] has recently described well the chaotic condition of our knowledge, or rather lack of it, and laid emphasis on the necessity of more careful study of the disease. We are to-day really proving the truth of words spoken by Haygarth in 1805, who said: "The term rheumatism has been applied without sufficient discrimination to a great variety of disorders, which, except pain, have but few symptoms that connect them together."

In the present paper a series of 110 cases which have occurred in the Johns Hopkins Hospital are reported. The clinical features will be especially discussed. A point of much importance is the relative frequency of the

1. American Medicine,, 1902, iv, 17.

disease. With the present confusion of our knowledge of the disease, any statistical figures seem to possess but little value. As some of the cases here reported suggest strongly that one type of the disease is very commonly taken for acute articular rheumatism, and as probably one other form of the disease—spondylitis—has usually been overlooked, statistics are only likely to lead to false conclusions. It probably occurs less frequently than true articular rheumatism, but the difference in number may not be as great as we have usually thought.

One point to which special attention will be drawn is the study of early diagnosis. How much we can do to prevent the later manifestations of the disease by recognition and treatment of the early conditions is difficult to say. Certainly we are in a position to try, whereas by making a diagnosis only when the damage is done, treatment then can only be palliative.

PATHOLOGY.

Of this we have but little accurate knowledge. With the etiologic feature unknown we can only discuss the morbid anatomy and not the pathologic basis. In the usual descriptions of the disease we have given only the later changes, many of which are degenerative. That the early acute conditions have received little attention is natural, for the material has been lacking. Patients rarely die of the disease, and certainly not in the early stages. Dr. White[2] has recently reported the result of an autopsy on a comparatively early case. It is to be hoped that no like opportunity for examination of such a case will be missed.

The two great views of the etiology—namely, the infective (either due to a specific organism or to a variety of organisms) and the neural—need not be discussed. The clinical features of this series seem rather to point toward an infective cause, although our cultural results have been negative.

---

2. Guy's Hospital Reports, lvii.

Some writers suggest that under the term arthritis deformans we have really two diseases. Whether or not this be correct, we can distinguish two types of cases, one in which the bony changes, especially hypertrophy, predominate, often termed the "osteoarthritic," and the other in which the joint structures other than the bony parts are especially involved, sometimes termed "rheumatoid." These are usually distinctly different conditions. Do they represent different diseases, or are they varying manifestations of the same disease? The answer is difficult at present, but there seems little doubt of the co-existence of lesions of both types in some patients. Thus, in patients with spondylitis usually regarded as being "osteoarthritic" we may find involvement of other joints with the condition regarded as "rheumatoid."[3] Whether the same etiologic factor is active in both, it is at present impossible to say.

Some reference should be made to the association of arthritis deformans and rheumatism. Some writers describe arthritis deformans as coming on after acute rheumatism. The two diseases may have occurred in the same patient, but it is not any more likely than that rheumatism and gout should have done so. If we see a patient in successive attacks of acute polyarthritis which we regard as acute rheumatism, until he comes back, perhaps, with a tophus, do we say that gout has followed acute rheumatism? If we are honest, we frankly admit our error in mistaking the nature of the arthritis. Why not the same in regard to arthritis deformans? When a patient comes with undoubted manifestations of this disease it seems more reasonable to consider previous acute attacks to have been of the same nature. The two diseases may have occurred in the same patient, but it seems more probable that the acute attacks were of arthritis deformans than acute rheuma-

3. The use of the term "rheumatoid" is to be regretted. Everything suggesting any association with rheumatism should, in my opinion, be eliminated. I have used the term "polyarticular" to designate the group in which these lesions especially occur.

tism. The large number of cases to be noted later support this view. It seems most reasonable to hold that arthritis deformans is distinct from acute rheumatism.

For clinical study it is necessary to divide the cases, and in this report the following classes are used:

1. Heberden's nodes.
2. Polyarticular form (excluding the spondylitis cases, which are in one sense polyarticular).
3. Monarticular form.
4. Spondylitis.

It must be understood that the lines between these are not always definite. A patient with spondylitis or Heberden's nodes may have general polyarticular involvement, although this mixing of the types is not very common.

The statistics for the whole series will first be given, and afterward each group will be discussed in detail. That the present series gives any guide to the relative frequency of various groups is not probable. This is especially to be kept in mind in reference to the cases with local involvement of the spine. The cases of general spinal involvement offer but little difficulty in recognition, but we have only recently learned to recognize the other cases, and therefore their absence in the records of previous years has been due to non-recognition. These cases occur especially in males, and doubtless future records will show a more equal distribution between the sexes than previous ones have done. It will be seen that in this series the males and females are equal, rather unusual in any large series.

It may be noted that no cases with Heberden's nodes only are included in this series. Two cases of the so-called "Still's disease" are included in the polyarticular group. These cases are somewhat doubtful, but it seems most reasonable to consider them as arthritis deformans.

GENERAL STATISTICS.

Incidence.—In the present series of 110 cases, 55 were males and 55 females. The white patients num-

bered 106, the colored only 4, 2 of whom were males and 2 females. The small number of colored patients with the disease is of considerable interest, especially in reference to the association of previous septic conditions as etiologic factors. The colored race is susceptible to the majority of infections, and especially gonorrhea. While in the hospital the proportion of white to colored is as about 7 to 1, the incidence of arthritis deformans is about 26 to 1. The negro is also apparently rather more prone to acute articular rheumatism, as has been elsewhere brought out,[4] the proportion in this clinic of white and colored with this disease being as 4.8 to 1.

Conjugal Condition.—Fifty-three of the patients were married, 41 single, and 16 were widows or widowers.

Nationality.—It is interesting to note the preponderance of the native-born. Ninety-six were born on this continent, and only 14 abroad. Of these, 8 were German, 2 Irish, 1 each English, Bohemian, Russian and Pole.

Occupation.—The largest number were engaged in housework of various kinds, namely, 47. Seven others were engaged in similar occupations, such as sewing, washing, etc. The next largest group comprised 24 cases; they were merchants, clerks, teachers, etc. There were 11 farmers and 10 engaged in laborers' and mechanics' work. The remainder were of no especial occupation. The proportion engaged in occupations involving special exposure to wet, damp, etc, is seen to be surprisingly small.

Age.—The age of patients on admission was as follows:

1 to 10	2	51 to 60	19
11 to 20	8	61 to 70	7
21 to 30	19	71 to 80	2
31 to 40	35	81 to 90	1
41 to 50	17		

The youngest patient was aged 4 (a case of "Still's

4. McCrae: The Journal A. M. A., Jan. 24, 1903.

disease"), the oldest 83, with Heberden's nodes and some changes in other joints.

Complaint.—The complaints made by the patients were very various. By far the largest group (36) complained of "rheumatism"; 23 complained of general joint symptoms, pain, stiffness and swelling; 20 complained especially of the pain; 10 of stiffness of the back, and the remainder of various things—6 of loss of power in the legs and difficulty in walking; 3 of sciatica; 2 of neuritis, etc.

Family History.—The family history is of interest. The difficulty in obtaining an exact description of an arthritis in the family history is well known. There was a definite family history of arthritis in 44 cases. Of these, 10 were undoubtedly arthritis deformans, 22 were given as rheumatism, 1 as gout, and in 11 the nature was unknown. Endeavor was made to find out if the disease developed earlier in the patients who had a family history of arthritis than in the others. The results are given in tabular form:

Age.	Age of onset with a family history of arthritis.		Percentage.	General percentage of onset in all.
1 to 10	7	=	16	11
11 to 20	10	=	23	20
21 to 30	9	=	21	20
31 to 40	7	=	16	22
41 to 50	4	=	9	9
51 to 60	4	=	9	12
61 to 70	2	=	5	3
71 to 80	0	=	0	2

One case was doubtful, and is not included in the first column, and two are doubtful in the second. The percentages are so nearly alike that no positive conclusion can be drawn, although the cases with a history of arthritis show a slightly larger percentage for the earlier decades. There was a family history of tuberculosis in 28 cases, and in 9 this was associated with arthritis.

Previous History.—Of the ordinary acute infections, measles is by far the most common, a history of it being obtained in 68 cases. Following this came whooping

cough in 31, mumps in 20, scarlet fever and malaria each in 18, chicken-pox and influenza each in 13, typhoid fever in 10, pneumonia in 7, diphtheria in 5, smallpox and erysipelas in 2 each. Tonsillitis in recurring attacks was noted only in 3 cases. There was one instance each of otitis media, bronchitis, pleurisy and carbuncles. There was a previous history of gonorrhea obtained in only 14 cases, or about 13 per cent. This, it will be noted, is in striking contrast to the percentage of 40, given in Dr. Stewart's[5] Montreal statistics. There was a history of syphilis in 4 and of chancroid in 1. Dyspepsia had been a prominent feature in 3 cases, and dysentery or diarrhea in 2. Four patients gave a history of nervous prostration; 2 insisted especially on their severe mental strain and worry for some years; 1 of great physical hardship, and 2 of working in especially damp surroundings. In regard to this latter, attention should be drawn to its apparent rarity in this series, as so many of the writers lay great stress on its importance as an etiologic factor.

Alcoholic History.—Twenty-seven of the patients had used alcohol, 6 described as heavy drinkers, the remainder moderate. This is in striking contrast to the figures given in the series of gout cases reported from this clinic, where practically all were alcoholics.

Pregnancy and Miscarriage.—Twenty-six of the female patients had borne children, and seven had had miscarriages, varying in number from 1 to 4. Five patients had both borne children and had miscarriages.

Onset.—This was apparently associated with some condition in 28 cases. The patients, at any rate, thought there was some association. In 7 cases the onset was associated with exposure to cold and wet; in 3 each the onset was apparently associated with injury, diarrhea or dysentery, confinement, an attack of influenza, and an attack of measles; 2 followed closely after typhoid fever; and in 1 each it was associated with the follow-

5. Stewart: Brit. Med. Jour., 1897, ii, p. 1225.

ing conditions: Very marked mental strain, constant physical strain, miscarriage and the menopause. The small number associated with exposure is to be noted. Our series of cases of acute rheumatism gave practically double the percentage. In one-half of all the cases the onset was before the age of 30, and in about one-third, before 20.

In the further study of the clinical types it is much more convenient to study each of the groups previously mentioned separately.

GROUP I. HEBERDEN'S NODES.

This clinical group requires but little discussion. Generally accepted as undoubted manifestations of arthritis deformans, anatomically, this must be considered as usually belonging to the osteoarthritic type previously referred to. No figures as to the relative frequency of their occurrence can be given. There can be little doubt that the general idea that they are rarely associated with the more severe manifestations of the disease in other joints is substantially correct. In the present series, Heberden's nodes occurred in association with manifestations in other joints only nine times among the 94 polyarticular cases. Of these 9, in 4 the other manifestations were slight; in 3, moderate, and in 2, severe. They did not occur in association with any of the cases of spondylitis. As to the idea of Charcot that patients having these nodes were especially prone to cancer, I have no figures to offer. Certainly, one sees the association not infrequently. One point of interest in this group is the association of these lesions which we term "osteoarthritic," with those in other joints which are undoubtedly more of the "rheumatoid type." That this does occur there can be no question. It certainly speaks for a possible association of the two types.

Frankly, we have to confess that of accurate knowledge as to Heberden's nodes we have little. It can be summed up by saying that these bony outgrowths at the

terminal phalangeal joints of the fingers rarely cause any troublesome symptoms, usually occur without the larger joints being involved, but may be associated with the other manifestations of the disease, although these cases are comparatively rare. In one particular they agree with the lesions in the other joints, namely, they are susceptible to injury. A blow on an affected joint is nearly always followed by some increase in the local symptoms, exactly as in the larger joints. Curiously enough, just as this was being written a patient came under observation with Heberden's nodes showing an unusual condition. She came on account of another disease. There was marked deformity of the terminal finger joints, and definite crepitus was obtained in some of them, an unusual thing in such cases in our experience.

GROUP II. POLYARTICULAR CASES.

This comprises by far the largest number of cases in this series. There are 92, to which may be added 2 cases of "Still's disease," although in the tables these are kept separate. These cases and their clinical features show many variations, but an attempt will be made to classify them, at any rate to some extent. As already said, the changes are largely of the "periarticular" kind, and the bony changes are secondary.

Onset.—The ages at onset of these cases are shown in the following table:

1 to 10	11	41 to 50	10
11 to 20	17	51 to 60	11
21 to 30	18	61 to 70	2
31 to 40	22	71 to 80	1

Method of Onset.—This is of especial interest. On studying the cases, one finds two fairly definite kinds of onset can be recognized, namely, a sudden and a gradual beginning. Of the 92 cases in this group, the method of onset was doubtful in 6, in 45 it was gradual (in 31 of these several joints being involved together at the beginning, and in 14 one joint, others following some time later); in 41 cases the onset was sudden, in 19 of

these in one joint, and in 21 in many joints about the same time. In one case the onset was in the joints of the neck. Especial attention is directed to the number of cases showing a sudden mode of onset. So many of the descriptions of the disease lead one to believe that the sudden onset is unusual, and that we see, ordinarily, a slow, gradually advancing arthritis. This point will be especially dealt with later. Of the 2 cases of "Still's disease," in 1 the onset was gradual in one joint, and in the other sudden in many joints.

The Joint First Involved.—This was as follows: 3, no definite history; 28, many joints involved; 3, the neck joint first involved; 5, the shoulders, 3 being only in one shoulder; 4, one arm; 1, an elbow; 3, a wrist; 1, a thumb; 1, a single finger; 1, both hands; 9, one hip; 10, one knee; 3, both knees; 1, knees and elbows both; 1, both ankles; 1, one ankle; 10, the feet; 3, the great toe; 2, other toes; 2, the joints of one leg.

The Character of the Attacks.—It is, of course, impossible in a disease such as this to definitely classify all the cases or the attacks. There is so much variation in the order of the joints involved, the character of the successive attacks, etc., that only an approximate classification is possible. Considering the course of the disease, perhaps three groups may be recognized. The first of these is the slow, progressive type, in which, after either an acute or a gradual onset, the symptoms slowly advance, joint after joint being involved. This may go on for a great many years. Of this class there are 44 cases. In sharp distinction comes the second class, those in which the attacks are acute and very commonly come on at intervals and at intervening times, perhaps for many years the patient may be practically free of any symptoms, although at length permanent damage is done. In certain of these cases the patient may apparently recover perfectly, or almost so, between the attacks, and it may be only after repeated acute attacks that permanent changes appear in the joints.

To this class belong 29 cases. There is a third class which shows in some ways features of both the first and second groups; namely, in that they had acute attacks, but with these there was a steady progress, a certain amount of damage being left each time. In this class there were 12 cases. The remaining 7 cases it was impossible to classify.

CLASS 1. *The Slow Progressive Type.*—It is not necessary to spend time in describing these cases. Beginning either acutely or slowly in one or many joints, the process gradually advances, perhaps sometimes aggravated by accidental conditions, to a greater or less degree of deformity. Many joints are usually involved, the changes being rather in the periarticular structures than in the bony ones, the adjacent muscular structures showing a marked atrophy, deformities resulting, and at the last, a condition of more or less helplessness. A typical case may be briefly quoted:

No. 87.—B. K., white, male, age 40. He was admitted complaining of contraction of the legs and inability to walk. His family history was negative. The history of his present trouble was that twenty-eight years before he began to have pain in the left hip. This soon became stiff and he had difficulty in walking. After six weeks he noticed pain in the other hip, and for a time had much difficulty in walking. He then got about for a time, but always limped. Four or five months later he had a second attack and spent over a year in bed, without any acute symptoms. At the end of this time the knees gradually became involved. After some months he managed to get about, although always more or less stooped, having to walk with the hands on the knees. After a time he had to give this up and take to a wheel chair. The knees became gradually fixed, and for twenty-two years he has not been able to walk. A history in which there was never any acute attack, but only a slow continued progress.

This case, of course, showed involvement only of the lower extremities. A second case may be quoted:

No. 26.—O. F., white, female, aged 40. She was admitted complaining of pain and deformity of the joints. There was a history of arthritis on both sides of the family, and the mother has arthritis deformans. The previous history was negative. Fifteen years before, after a wetting, she com-

plained of some pain in the left arm, which was thought to be rheumatism, was not severe, and did not last long. After this she was free of symptoms until ten years ago, when she began to have pain in the feet, especially after walking. There was no swelling, and the pain was not especially in the joints. At this time she began to notice some changes beginning in the finger joints. These gradually progressed, and for five years she had some general pains and deformity, especially of the feet and hands. During this time her general health varied. It was usually rather bad. She was very nervous. Five years ago her symptoms became very troublesome. The knees were involved, swollen and tender; pain appeared in the neck, and the temporomaxillary joint was involved. After this there was gradual swelling of the ankles, elbows and shoulders. For six months past she had pain and was almost helpless, requiring to have help in putting on her clothes, taking her meals and getting about. There was nothing acute in the symptoms at any time.

Such cases might be quoted at length, but to no purpose. The type is perfectly familiar.

Of the second group, namely, the acute, two illustrative cases may be quoted:

No. 48.—M. W., white, female, age 60. She was admitted complaining of rheumatism. She had a family history of arthritis. Her previous history was negative. At thirty-five she had an attack of acute arthritis, with swelling and pain in many joints. She was laid up in bed for two weeks. Afterward she apparently recovered completely. She had a second attack much like this at forty-five, after which she was practically well. The menopause occurred at fifty-three. Fifteen months ago she began to have pain in the great toe of the left foot. After this the knees, shoulders, elbows, wrists, and hands were all involved. The changes in the joints were more or less permanent, and she now has difficulty in getting about. She herself had noticed that the joints, although swollen and painful, were not red, and that she had no fever.

This is a fairly common history in these cases, recurring acute attacks leaving little damage, but finally permanent changes occurring. A second case in a boy of 13 may be quoted:

No. 53.—E. S., white male, age 13. He complained of swelling and pain in the feet and legs. His family and previous history were negative. His first attack of arthritis was at seven years. This lasted only for a short time, but was accompanied by severe pain. Following this he had eight or ten attacks, all acute, the first one lasting only three

days. The one before admission had lasted for two months. Between the earlier attacks he would be perfectly well in every way, but after the later ones it was noticed that a certain amount of stiffness persisted. The last attack was ten months before admission. He had gone to bed well. The next morning on rising he found both ankles so swollen that he could not get his boots on and he was unable to stand. Three months later the wrists became involved suddenly and remained so. On admission the involvement was in many joints.

Many histories of features much like these might be quoted, but it is unnecessary. They all serve to emphasize, however, the comparative frequency of this type of the disease. It is such histories that have suggested previous attacks of acute rheumatism as being common before the development of arthritis deformans. That all these attacks were of the one disease there seems little doubt. In the majority of these cases there is little difficulty in distinguishing them from acute rheumatism. This will be considered later in the section on diagnosis. But a point which seems worthy of emphasis is the frequency of these acute attacks. Nearly one-half of the cases of this group showed these acute manifestations at one time or other. Of the 32 cases in which there was a history of previous acute attacks after which they were practically free of symptoms, 14 had one such previous attack, 2 had two attacks, 4 three attacks, 4 four attacks, 2 five attacks, and 1 ten attacks.

There were 33 cases in which an intercurrent condition was thought to have determined the onset of another attack or further symptoms. In 8 this was an injury of some kind, usually to one joint, and followed at once by acute symptoms in it. Sometimes after this other joints followed. In 6 a wetting or exposure, in 5 an attack of dysentery or diarrhea seemed to have determined a fresh outbreak, in 2 an attack of influenza, in 2 confinement (repeated in successive pregnancies), in 2 a miscarriage, in 2 a marked shock, and in 1 each an attack of gonorrhea, an attack of measles, hot bathing, excessive fatigue, menstruation and a chancroid. Some

of these may be only coincidence, but in the majority the association seemed definite.

CONSIDERATION OF THE SYMPTOMS IN DETAIL.

In the discussion of these, especially the features pertaining to the joints, we have to keep in mind the more acute forms and the chronic deformities. With the latter especially we are familiar, and they require but little description. Along with these goes the muscular atrophy which is so common. To consider first the features in the joints:

The Cervical Vertebræ.—These require especial mention quite apart from the condition of spondylitis, because at some time or other in the polyarticular form it is not uncommon to have the neck involved. This was the case in 29 out of the 92 cases. This involvement we have often found to be transitory. There may for a time be pain complained of on moving, motion is much restricted, but on ordinary examination little is to be made out. Sometimes there is some deformity, the head being held definitely to one side. Judging from our cases, the condition is rarely permanent, many of the patients, especially in the acute attacks, having marked symptoms, which disappear completely as they improve. In very few of our cases has there been any permanent stiffness, apart, of course, from the cases of spondylitis.

The Temporo-Maxillary Joint.—This is involved comparatively frequently—in 22 cases of this series. Involvement is often unilateral and transient, but there may be all variations from this up to complete ankylosis, so that the patient may require to have teeth removed in order to be fed. This, however, has been rare in our cases. In the majority of instances there has been no permanent disability. In a few cases there was almost complete ankylosis and afterward fair recovery. One patient had teeth removed at one time, and later was able to open the mouth to a moderate extent. Involve-

ment of this joint should be considered an important point in the diagnosis of a doubtful polyarthritis.

The Sternal Clavicular Joint.—This was involved in 6 instances. There was usually pain, sometimes slight swelling, and complaint of any motion involving the clavicle. It apparently is not a serious condition, and rarely causes any great discomfort.

The Shoulder Joints.—One or both shoulders were involved in 50 instances; of these, both shoulders in 39 cases; the right only in 8 cases; the left only in 3 cases.

In the acute attacks the patient usually complains of pain and limitation of motion, especially shown in attempts to raise the hand toward the head. It is very common to see them able to raise the hand to the level of the shoulder, but no higher. When the joint condition becomes more chronic there is usually the continuation of pain, some stiffness on handling, often definite limitation of motion, and very commonly crepitus. Fluid in the shoulder joints seems to be comparatively rare. Later there is often a certain amount of muscular atrophy of the shoulder girdle muscles, which, with the disability, often gives a very characteristic picture.

The Elbow Joints.—These were involved in 43 cases: Both elbows in 31 cases; the right only in 4 cases; the left only in 8 cases. In the acute attacks the elbow is usually held in a flexed position, there is often some swelling, and not infrequently a certain amount of involvement of the muscular tissues above and below the joint. This seems often more marked about the elbow than the other joints. In one case the elbow joint was opened during a moderately acute condition. The patient was thought to have gonorrheal arthritis. The muscles were found to be slightly edematous, and there was some fibrin between the muscle fibers. There were some small areas of hemorrhage. The joint contained some viscid yellow fluid. The synovial membrane showed hemorrhage and had a gelatinous appearance. In the later stages the joint may be fixed in flexion or

partial flexion, but the thickening about the joints rarely advances to the extent that it often does about the knees.

The Wrist Joints.—These were involved in 46 cases: Both wrists in 36 cases; the right only in 5 cases; the left only in 5 cases. The changes in the wrists are often very characteristic. Only occasionally is there effusion into the joint (noted in one instance in this series), but there is often marked swelling above and below the joint, especially below. As a result of this the usual outlines of the wrist become completely lost. In the later stages we may find extreme polyarticular thickening and a certain loss of motion, although this is usually not so marked as at the elbow. Crepitus is very often obtained.

The Hands.—There was involvement of the metacarpal-phalangeal joints in 41 cases. The changes in these joints are usually very characteristic. In the acute attacks there may be a certain amount of redness, swelling and tenderness. As this subsides, a variable amount of thickening is left around the joints. In the later stages the thickening is permanent, mobility is lessened, there is usually flexion of the joints, and, not uncommonly, some subluxation. It is not uncommon to find some of these joints involved and others free. In six other cases of the series there is only a note that the hands were involved, which probably means these joints.

The Fingers.—These were involved in 52 cases. The thumbs were only involved in 7. The changes in the fingers were often very characteristic. In the acute attacks there may be but little visible change. There is some swelling and limitation of motion, but no great deformity. Subsequently, the thickening about the joints becomes more marked, and the typical "pod-shaped" or fusiform finger is produced. In the later stages we have a great variety of deformities. A common form is a flexion of the first phalanx with hyperextension of the terminal ones, but all the joints may be partially flexed, or only the terminal one be hyperextended.

With this one may have also lateral deflection, especially of the terminal joint; and in some cases subluxation may result.

There are several features associated with the hands worthy of note. The patients are rarely able to "make a fist." There is very commonly ulnar deflection of all the fingers, which probably means only the position of greater ease. There are some other features which may possibly not be associated so much with the joints as with other structures. One of these is sudden weakness of the wrist joint, so that the patient drops articles which are being held. This is not an uncommon occurrence, sometimes happening in the earlier stages. Another point is the complaint of pain about the ball of the thumb, or on the ulnar surface of the wrist. This may be found in certain instances quite apart from any active arthritis, and it seems worthy of being noted. In certain cases one notes atrophic changes about the hands. In others the nails are ribbed, or show coarse markings. The skin, in common with that of other parts of the body, may be glossy and shining.

The Hip Joints.—These were involved in 31 cases: Both hips in 25 cases; the right only in 3 cases; the left only in 3 cases. It is not uncommon to have complaint made of pain in the hip joint during an acute attack, but on examination nothing very definite is to be made out except tenderness. In the later stages there may be, as in other joints, more or less permanent deformity. The joint becomes fixed, often with the thigh slightly flexed and more or less abducted. The changes are often not as marked as in the cases with monarticular disease of the hips or spondylitis.

The Knee Joints.—These are by far the most frequently involved in the cases of this series, there being a total of 80 cases. The next most frequent of the single joints is the shoulder. The figures are: Both knees in 65 cases; the right only in 7 cases; the left only in 8 cases. Effusion occurs in the knee joint with considera-

ble frequency, this being quite marked in 14 instances.[6] The knee joints in the acute attacks often show nothing very characteristic. There is some redness, swelling and tenderness. In later stages the joint commonly becomes somewhat fixed, and there is very often crepitus and marked thickening in the polyarticular structures. Along with this frequently goes marked thickening of the capsule. This can be readily appreciated by running the thumb up and down the inner surface of the joint opposite the interval between the two bones. A thickening of the capsule can thus be very readily perceived, and the thickened folds readily felt. One is often deceived into thinking that there is marked enlargement of the bony parts of the knee joints, whereas this appearance is due principally to the periarticular thickening and muscular atrophy above and below the joint.

The Ankle Joints.—These were involved in 48 cases: Both ankles in 37 cases; the right alone in 5 cases; the left alone in 6 cases. The ankles show often much the same features as the wrists. There is considerable swelling about the joint, and there is stiffness and restriction of motion, with but comparatively little change in the joint itself.

The Feet and Toes.—There was involvement in some of the joints of the feet in 11 cases; of the toes in 10; of the great toe only in 6. The disparity between the involvement of the fingers and of the toes is very striking. Notice should be directed to the cases having involvement only of the great toe among the joints of the feet. In a doubtful case this suggests gout, but the changes in the other joints may be characteristic.

The Symmetry of the Joint Involved.—The majority of writers are very fond of laying emphasis on the symmetry of the disease, but, as will be noted from the figures given before of joint involvement, in a considera-

6. It is, of course, often difficult to be certain of the presence of small amounts of fluid in a joint. Probably it occurs much more frequently than figures generally indicate. In all these cases the presence of fluid was established beyond a doubt.

ble number of instances only one joint was involved. For example, in 11 out of 50 cases of shoulder joint involvement, the disease was unilateral; in 12 out of 43 cases at the elbow joint; in 15 out of 80 in the knee joint, etc. The total number of instances of involvement of the joints of the upper extremity was 238, and of the lower extremity 186. The larger joints of the leg were involved 159 times, and those of the arms 126 times. The preponderance of joint involvement was markedly in the upper extremity in 4 cases, in the lower extremity in 12. This preponderance is, of course, analogous to the greater frequency of the involvement of the large joints of the leg. In only three instances, however, were the joints of one side of the body involved to the exclusion of the other side. One pair of joints only were involved in 3 cases, in 2 the knees, and in 1 the wrists.

How satisfactory the explanation sometimes given may be (namely, that the joints most involved are the ones most in use), there can be little doubt that, as a general rule, the large joints of the legs suffer more than the large joints of the arms. The hands suffer more than the feet. In reference to the association of the polyarticular forms with Heberden's nodes, the figures have already been given. Of the cases with spondylitis there was involvement with the other joints in 8 cases, in 4 of which there were multiple joints; in 1 in the hands; in 1 in the shoulders; in 1 in the knees and hips, and in 1 in the knees, hips and ankles.

Effusion.—The fluid obtained by aspiration is usually somewhat turbid, and contains a certain amount of granular material, with polymorphonuclear leucocytes. We have never found a hemorrhagic exudate.

Muscular Atrophy.—This is present in the majority of cases, but it is well to remember that in certain of the acute forms with a short duration there may be but little atrophy until after successive attacks. This is not invariable, for even after a short duration of symp-

toms in some cases there may be quite marked atrophy. The significance of this it is not the purpose to discuss here, nor to what extent it suggests a diseased condition in the central nervous system. The atrophy is often characteristic and striking, especially with the appearances in the joints. This is often true of the hands when the wasting of the interossei gives a very typical picture. The rapid onset of this sometimes gives a clew to the diagnosis in a doubtful case.

General Joint Features in the Acute Attacks.—Reference may be made to these, especially as there is so much difficulty in distinguishing the condition from acute rheumatism. There is usually a polyarthritis, but rarely any succession of joints becoming involved and then clear, as in rheumatism. A joint when once attacked is rarely free until the attack is over. There is usually considerable pain, moderate swelling, which may extend above and below the joint (especially in the elbows, wrists and knees), at times redness, but not as marked as in rheumatism, sometimes some heat, and rarely extreme tenderness. If with these features there be involvement of the joints of the hands or fingers, of the cervical vertebræ or temporomaxillary joints, the diagnosis is fairly certain. With these local conditions there is usually not high fever (often surprising when the degree of arthritis is considered), but a rather rapid pulse rate. There is rarely endocardial involvement. To sum up, a polyarthritis which does not shift about, slight fever and a rapid pulse, are the most striking features. As time goes on the fact that the joints change very little, the arthritis persists and the usual rheumatic remedies are of little avail, all help to decide the diagnosis. After the subsidence of the acute features the persistence of thickening about the joints is a most suggestive feature.

Temperature.—Of this there were records in 86 cases, the remaining patients not being in the hospital long

enough to obtain a complete record. Of these the figures are as follows:

Normal	15
An average of 99	28
An average of 99 to 100.0	21
An average of 99 to 100.5	5
An average of 99 to 101.0	10
An average of 99 to 102.0	1
An average of 99 to 103.0	1
An average of 100	1
An average of 100 to 102.0	1
An average of 101 to 102.0	1
Irregular	2

Of the two irregular cases, in one the temperature varied from 99 F. to 105 F. This was associated with a general pneumococcus infection and death. In the other case the patient had a severe shaking chill, and the temperature arose to 104 F.; the only instance in the series where a severe shaking chill was noted.

These figures of the temperature records are of considerable importance. It will be seen that only fifteen cases, or 18 per cent., had a normal temperature, and that the great majority, fifty-nine, had a temperature averaging between 99 F. and 101 F. This is a point of considerable aid in the diagnosis. The majority have fever, but it is usually not high.

The Circulatory System.—There are notes of the cardiac condition in only 74 cases. Among these the heart was perfectly clear in 55. There was a soft systolic bruit, apparently not organic, in 10. In 2 there was marked irregularity, and in 7 an organic lesion. Among these latter cases there were 3 of aortic insufficiency, 2 of mitral stenosis and insufficiency, and 1 each of mitral insufficiency and mitral stenosis. In some of these the condition was associated with marked general arterial sclerosis, and probably the cardiac condition was due to this.

Of much greater significance are the records of the pulse rate. Of the 78 cases in which there were long enough records to note, the figures were as follows:

70 to 80	9	100 to 110	19
80 to 90	17	110 to 120	7
90 to 100	24	120 to 130	2

It will be seen that in two-thirds of the cases the pulse

rate was above 90. This may be not only with the acute attacks, but also in the conditions characterized by more or less slow advance. We have found it of considerable aid in the diagnosis, especially in association with slight fever.

Urine.—This showed practically nothing abnormal. In 83 cases it was clear in 64, contained a very slight trace of albumin in 9, and albumin and casts in 10. Sugar was found in one instance. Determination of the total excretion of uric acid was made in a number of cases, but always with the finding of the normal amount.

Glandular Enlargement.—Of 33 cases of which we have careful records there was general glandular enlargement in 13, definite enlargement of some of the glands in 4, and no general enlargement in 16. This condition would probably have been found in many other instances had it been carefully looked for. In the majority of cases, at any rate with acute features, the glands in association with the affected joints are enlarged. This is worthy of note as supporting the view that the condition is an infective process of some kind.

Spleen.—Of 39 cases where there are careful notes, the spleen was found enlarged in 4, and in 35 no increase in size could be made out. This is a point of considerable interest, especially in association with the cases of "Still's disease," in which the enlargement of the spleen is such a marked feature.

Subcutaneous Fibroid Nodules.—Of these there are the surprisingly large number of 7 cases, and one additional doubtful case, in which the enlargement seemed to be in association with the tendon. Regarding these nodules, there is no doubt that they are found exactly in proportion as they are looked for. In this clinic careful search is usually made for them, and in all these cases their presence was confirmed by Dr. Osler's note. These figures give a percentage of 7.4, which is very much larger than in the instances among our cases of acute rheumatism (namely, 1.5 per cent.). The opinion

is now fairly general that these nodules are not essentially rheumatic, although some writers would have us believe that they were absolutely diagnostic of a rheumatic condition. Of special note is the age of the patients in whom they were found. One was 27, one 33, and all of the others over 50 years of age. In rheumatism these nodules are almost always found in children.

Pigmentation.—Marked pigmentation was noted in 8 cases of the series, but it is doubtful if this represents the total number, had it always been noted. Some writers, especially Spender, have laid emphasis on the occurrence of brown pigmented areas about the forehead, face and forearms, especially as being often diagnostic. If one follows for a time the general incidence of these pigmented areas among all patients they seem to me to be about as frequent in other conditions as in arthritis deformans.

Blood.—The average percentage of hemoglobin in 33 cases was 70.6, and the red cells in 29 cases, 4,468,000. These figures seem rather high when one remembers the very frequent pale, anemic look of these patients; but the pallor is often more striking than the actual anemia.

The average leucocyte count in 33 cases was 7,600. Of these in only 9 was the count over 10,000. In two of these the leucocytosis was probably due to other conditions. In one with an acute pleurisy the leucocytes arose to 18,000; in the other the count of 15,000 was associated with a general pneumococcus infection. These two counts are not included in the average. Of the remaining 7 cases it is interesting to note that in only 2 were the counts taken during acute attacks, rather a surprising result. In 6 cases the leucocytes were 5,000 or lower. Full differential counts were made in 8 cases. These gave an average of: Polymorphonuclears, 76 per cent.; small mononuclears, 14 per cent.; large mononuclears and transitionals, 8 per cent.; eosinophiles, 1.5 per cent.; mastzellen, 0.5 per cent. These are practically normal findings.

Reflexes.—The study of these is of interest, especially as some writers, R. L. Jones,[6] for example, attach some importance to them in the diagnosis. In the cases of this series there are not enough complete reports of the reflexes to draw any conclusions. But the general rule seems fairly well established that the reflexes in association with affected joints are usually increased. This was very evident in some of our cases where the involvement was asymmetrical. On the side affected the reflexes were exaggerated, but normal on the sound side. Whether the explanation lies in the changed local condition or alterations in the cord, is difficult to say. In addition to the deep reflexes, the superficial ones are often much exaggerated, which we have found especially true of the cremasteric reflex. This rather speaks for other than a local cause.

But the findings are not constant. The cases are so variable and the conditions so different that this may later be easily explained. Practically, the reflexes in different cases have been found exaggerated, normal, or decreased, but rarely absent. Ankle clonus was obtained in a few instances, but we have never obtained a definite extensor response to plantar stimulation.

How strongly the conditions of the reflexes speak for spinal cord involvement it hardly seems yet possible definitely to say.

Unusual Features.—These require no special mention. They comprise: Pleurisy, 2; severe diarrhea, 2; chill, 1; jaundice, 1; abdominal pain, 1; mental features, 1; purpura, 1; enlarged thyroid, 1; bronchitis, 1; paralysis, 1. The case with severe abdominal pain is of interest, as there have been cases with like features reported both in acute rheumatism and gout.

PATHOLOGY.

This is again one of the doubtful points in the disease. Until we are certain of the etiology we can determine

6. The Lancet, Dec. 27, 1902.

the morbid anatomy only. The essential element in our knowledge of the pathology is lacking. It is not the purpose here to discuss the various opinions at length. The two principal views are that the disease is (1) due to an infective agent (either a specific organism or a variety of organisms or their toxins) or (2) of neural origin.

For the infective view there are many suggestive points. Here, of course, only the polyarticular form is discussed. The mode of onset, often sudden, with involvement of many joints, the fever, increased pulse rate, are all in favor of an infection. The subsidence of these after a time, often to recur in subsequent attacks—the interval perhaps having been free of symptoms—and usually first or last the appearance of permanent changes about the joints are all important. In some patients there is permanent change dating from the first attack, in others this may only occur after repeated attacks. These seem comparable to the condition often seen in the endocardium of rheumatic patients. A first attack may leave the valves permanently damaged, or only more liable to involvement in subsequent attacks.

Could we determine a definite organism to be associated with the disease, the matter is settled. In this series we have never succeeded in obtaining any results from cultures, in spite of the greatest care in following the methods given (for example, by Bloxall). If we hold the view that many organisms (such as those of influenza, gonorrhea, etc.) are etiologic factors, there are many suggestive facts to support this. There seems no doubt of the association of attacks or exacerbations with various infections such as influenza. This may mean only lowered resistance, for we find this an important factor. Ill health from any cause, anemic or depressing conditions, may all apparently precipitate an exacerbation or another attack. In reference to the association with, for example, previous gonorrhea, it has already been noted that the disease is here very uncommon

among the colored race, among whom gonorrhea is almost of general occurrence.

Some features are usually referred to as being much in favor of a neural origin, such as the rapid muscular atrophy, the curious association of alteration in reflexes, such as between the gluteal and plantar, etc. Of the rapid muscular atrophy there can be no doubt. It may occur long before there is any possibility of disuse producing it. After a very prolonged attack of acute rheumatism there may be considerable atrophy, but rarely with the rapidity and certainly not with the certainty of its development in arthritis deformans. The association of the reflexes often follows their segmental distribution, as may also the curious localized distribution of the lesions in the hands. Thus the middle and ring fingers only may be involved, or show special changes. In another case the thumb and first finger may show the same association. These may speak for primary spinal cord involvement, but the explanation suggested that they may be due to changes due to toxic substances seems worthy of note. The changes found in the posterior columns of the cord do not seem necessarily primary. We know that they may be found with any chronic toxic condition, as, for example, diabetes or pernicious anemia. May not the chronic cases of arthritis deformans with suggestive nervous manifestations possibly belong to the same class?

A point worth noting is the occurrence of enlarged lymphatic glands in many of the cases, nearly half of these where we have complete records. In the so-called "Still's disease" they are very marked. This is no more than we might have expected, if this be regarded as a form of arthritis deformans. The glandular system of children reacts more markedly to toxic absorption than that of adults. This glandular involvement seems to support the view that we have a toxic condition, probably due to some infection.

While the whole question must as yet be left open, the

evidence seems to point more to the infective theory. Toxemia, secondary to infection, explains some, at any rate, of the nervous symptoms.

Morbid Anatomy.—Here again, in the majority of descriptions, we find only the late changes described. Opportunities for autopsies in the early stages are very rare. To describe the late changes throws no light on the condition. The majority might come from any long-continued degenerative joint condition. It is these that the majority of the writers have given as the pathologic anatomy.

One of the most important of recent observations has been made by Dr. Hale White, who had an opportunity to examine a comparatively acute case postmortem. In a proximal interphalangeal joint with marked changes he found that the most marked alteration was in the synovial membrane. This showed the formation of new fibrous tissue, and evidence of both recent and chronic inflammation. The membrane on the cartilage opposite the thickened fringe showed thickening, and the formation of fibrous tissue. The cartilage beneath this was thinned and eroded, and in places the erosion had gone through the cartilage. In connection with these areas there were foci of inflammation in the bone, but these changes were slight in comparison with those found in the synovial membrane. The synovial fringes in the knee joint showed no bony deformity, only the usual "thinned" condition of the ends of the bones so characteristic of these cases.

The only case of this series coming to autopsy affords but little information, so far as concerns the morbid anatomy of the disease. The patient was admitted with a general pneumococcus infection, with which was a pneumococcus arthritis. He had symptoms of arthritis deformans for over two years. The joint changes had gone on to erosion and eburnation of the bones. Each knee joint contained about 100 c.c. of purulent fluid. The synovial membrane was much thickened and inject-

ed, and there was evidently much overgrowth of connective tissue. Sections of these showed the surface covered with an exudate consisting of granular material and of polynuclear leucocytes. Below this was a layer of flat endothelial cells which merged into underlying fibroblasts, under which were many blood vessels and polynuclear leucocytes. Next to this layer was much fibrous tissue. These all seem to speak for inflammatory changes more than degenerative, at any rate in the early stages.

The findings—few though they be—all speak for an inflammatory condition at the onset. Degeneration comes later, but the beginning seems rather due to inflammation beginning in the synovial structures. This suggests infection, rather than neural causation.

Some writers have laid stress on various septic conditions, such as pyorrhea alveolaris. This may have some causal association, but its occurrence is so nearly universal as to throw doubt on its importance.

DIAGNOSIS.

Undoubtedly an essential thing in a correct diagnosis is a knowledge of the fact that arthritis deformans often occurs as an acute polyarthritis, with a sudden onset, and that unless care is taken the condition is especially apt to be regarded as acute articular rheumatism. Here, as is usually the case, knowledge of the possibilities is an essential to correct diagnosis. It may be taken for granted that the advanced stages with the marked characteristic deformity are not likely to be mistaken, but of what value is diagnosis then when the possibility of preventing these later changes is the hoped-for thing? Practically, the study of the diagnosis will be confined to the early stages. The more acute forms have to be diagnosed, especially from acute articular rheumatism, acute gout, acute polyarthritis from other causes (for example, septic conditions, etc.), and gonorrheal arthritis.

Acute Articular Rheumatism.—There are several fea-

tures that give one help in the diagnosis: the first, it may be said, is the knowledge of the two diseases. The polyarthritis of arthritis deformans rarely shifts from joint to joint. When the joint is once attacked, it rarely clears up suddenly. One seldom sees the rapid disappearance of arthritis in a given joint, which is so characteristic of rheumatism. The joints rarely show the severe local tenderness of rheumatism. There may be redness, but it is rarely extreme. The swelling is often marked, but in many of the joints it is distinctly more in the surrounding structures than in the joint itself. There may be effusion, especially in the knee joints. Certain joints are often involved which are rarely concerned in rheumatism, especially the joints of the neck and the temporomaxillary joint. The involvement of the fingers is often especially characteristic. We may find that only two fingers are involved, very commonly the middle or ring fingers, or it may be the first finger and the thumb. There is a curious appearance often given by the fingers, really the early stage of the "fusiform" condition. This we have found of considerable help. The temperature is usually not very high, and it may seem low in proportion to the amount of arthritis. The pulse is nearly always above normal. If this persists after the subsidence of the acute features, it is a very suspicious factor. The enlargement of the lymph glands, especially those associated with the affected joints, speaks for arthritis deformans. The absence of cardiac involvement and the usual lack of any help from the use of salicylates, is often an important point. Another suggestive fact is the common rapid muscular atrophy. Undoubtedly in prolonged cases of rheumatism one sees considerable atrophy, but it rarely appears with the rapidity that it does in arthritis deformans. The early atrophy of the interossei muscles is especially to be noted. The exaggeration of the reflexes is a point to be kept in mind. Then, too, the case is apt to drag on, the temperature perhaps remaining at 99 F., or slightly

over it, the pulse rate increased, and the joint symptoms persisting. This is always suspicious, and in case of a diagnosis of acute rheumatism, should always lead to a careful re-examination of the case. If after the acute symptoms are over joint deformity or alteration persists, one should always reconsider a diagnosis of acute rheumatism.

Acute Gout.—This is not likely to be a frequent cause of difficulty, but it is a possibility. The common alcoholic history in such patients, the attacks usually clearing up rapidly and leaving little damage in the joints, and the results of uric acid estimations, should all prevent error.

Gonorrheal Arthritis.—This is likely to give difficulty in diagnosis only where several joints are involved. If one cultivates the habit of always keeping gonorrheal arthritis in mind, and searches carefully for gonococci, there is not likely to be much possibility of mistake. In all doubtful cases of polyarthritis one should cultivate a "suspicious" frame of mind toward the diagnosis of acute articular rheumatism, and make it not first but last, and after other conditions have been excluded.

From various unusual joint conditions there would seem to be little difficulty in distinguishing arthritis deformans, and this group does not require discussion.

One is much impressed in going over the histories of our cases with the very common diagnosis of acute rheumatism made in the previous attacks. Often there are little facts mentioned which are enough to clear the diagnosis. Thus, several patients have said that their doctor commented on the fact that they had acute articular rheumatism with very little fever, or that they had a great deal of thickening left about the wrists or hands after their rheumatism. It is a good safe rule always to hold grave doubts as to the correctness of the diagnosis of acute rheumatism when there are permanent changes left in the joints after the acute attack is over.

Regarding what may be termed early minor manifesta-

tions of the disease, I do not know that any one has written better than Dr. Spender, in the article in "Allbutt's System of Medicine." These may be merely vague pains about the ankles or wrists, or the patient may complain of pain in one joint perhaps associated with some injury or a sprain. There may be some weakness of the wrists or hands. There may be curious neuralgic pains about the joints. As Spender says, these should always attract careful attention, and a thorough examination should be made. Of some of the other special points in the diagnosis to which Spender drew attention, note may be made. He emphasizes especially the rapidity of pulse. This, as we have already said, seems an important point. The various areas of brown pigmentation, especially on the face, neck and forearms of which he speaks, have not been of any especial assistance, and there would seem considerable doubt of their distinct association with the disease. The coldness and sweating of the hands do occur frequently, but it is probably more often associated with neurotic conditions, by no means a rare accompaniment of arthritis deformans. As Spender says, the diagnosis of rheumatism, muscular rheumatism, sprains, etc., are all pitfalls into which we are too apt to stumble.

TREATMENT.

In the discussion of this matter we are again met with the fact that there are two great groups of cases, the early and the late. The treatment in the first may undoubtedly be of great help as a preventive measure of farther damage. In the latter condition it can be only palliative, although even here much good may be done. We may first discuss the treatment of an acute attack. Patients are better at rest, and probably it is well to keep them clothed in flannel. If there be much fever the diet should be liquid. Ordinary attention should be given to the bowels. For remedies for the pain we have found that salicylates are of comparatively little use. Antipyrin in doses of 5 grains is often

effectual. Guaiacol is highly recommended by some. Small doses of codeia may be used; morphia seems to be rarely required. Of local measures to the joints the ice bag sometimes gives considerable relief, or the wrapping of the joint in cold compresses surrounded by oiled silk. These may be kept on constantly. Oil of wintergreen or lead and opium lotion applied locally is of some help. The hot air baking very often gives relief to the pain. Gentle massage may be of use. In spite of all measures, in some cases the patient is but little relieved. After an acute attack, whether the patient be absolutely free of symptoms or has some damage left in the joints, the great principle of treatment seems to be keeping up the nutrition. The patients should be out of doors as much as possible. They should be given all the nourishment they will take, and this of a general kind. The mistake is too often made in considering these patients to be rheumatic or gouty, and meat is almost totally cut off. On the contrary, a full meat diet seems especially helpful. They should be given nourishment between meals and at bedtime, and every effort made to improve their general condition. With this, of course, local derangements are to be carefully treated. Carious teeth and pyorrhea alveolaris should receive attention. Any stomach or bowel disorder should be remedied, and every endeavor made to keep the patient under the most favorable conditions. General massage is usually helpful. Concerning the various forms of bathing no general rule can be laid down. In England there can be little doubt of the benefit of such waters as at Bath. In this country, while some patients are doubtless helped by the bathing, many others are made distinctly worse. That this is due to too prolonged hot bathing to the point of depression may be possible, but our cases rather suggest that great caution should be exercised in advising, at any rate, any severe course of baths.

The local treatment of the joints is very important. First comes massage, which in the beginning ought to

be given by an assistant. But as a general rule later on the patient should be encouraged, as far as possible, to rub himself. There is usually the double benefit to the joint rubbed and to the hand employed. The hot air treatment is especially useful in many of these cases. The oven may usually be heated to 350 F., with perfect comfort to the patient, the importance of short applications being always kept in mind. Probably the majority of cases are helped most by periods of only twenty to thirty minutes in the hot air bath, and periods of an hour and an hour and a half are undoubtedly, in many cases, positively harmful.

The question often arises as to the amount of use that should be made of the affected joints. Leaving out the acute attacks, as a general rule, the more the joints are used the better. The mistake is often very serious when the patient is put to bed and any attempt at using the joints discouraged. The benefit of use is well shown by the temporomaxillary joint, which, although frequently involved, relatively rarely shows permanent damage. These measures may result in great improvement, even in a far advanced case, as, for example, in Case No. 70, that of a woman who for eighteen months had been bedridden, had been unable to feed herself, and who had some teeth pulled in order to be fed. After three months of treatment she was able to walk moderately well, could dress and feed herself, and open the mouth fairly well. Such cases are, of course, not common, but they serve to show that the outlook is not always without hope. There are two things necessary, probably both in the physician and in the patient. These are perseverance and patience. There are probably few cases, even of advanced disease, that can not be at any rate helped by persistent, patient, systematic treatment.

By far, however, the more important group, as regards treatment, are the early cases. As has been stated, too often these are regarded as rheumatism for long periods, until the damage is done. In these, when recognized

early, the most important element in the treatment seems to be good nutrition. General good hygiene, lots of fresh air and sunlight, and an abundance of good food are essential. Great care should always be taken to be on the outlook for the slightest manifestations of a return of the symptoms. Every slight infection should be regarded seriously. We are unable to say how successful early recognition of the disease and persistent care may be in preventing late attacks and permanent damage. At any rate, the patients should have the chance.

In addition to all the measures previously spoken of, there seem to be two drugs which are especially useful. These are the syrup of the iodid of iron and arsenic. They may be given alternately or combined, but in any case their administration should be kept up over long periods of time, for months, or if necessary, even years, at intervals.

Surgical Treatment.—This in some cases holds out a hope of benefit. In certain of the cases with marked contractures, the tendons may be divided and the limbs straightened. In other instances, under an anesthetic, a bent limb can be forcibly straightened and put up in plaster for a few days, but it can not be kept too strongly in mind that the best treatment for these cases of contractures is the preventive one. It is not, however, always in our power to carry this out, as the patients frequently delay consultation until permanent contractures have been established. Of excision of the joints we have had little experience, but it is undoubtedly of use in certain cases. It is probably most likely to be useful in the knee or elbow joint. It may serve to give a more useful limb. In some instances, too, the excision of the joint seems to be justified on account of pain. We see patients suffering with constant severe pain which nothing relieves; excision of the joint frequently gives great relief and the patients are left with about as good a limb as before. In some cases operations on the hip have been of considerable use. It must be said, however,

that as yet we have hardly enough statistics on which to draw any definite conclusions on the value of surgical intervention.

It can not be too strongly emphasized that in this and so many other diseases the great requisite to intelligent treatment is proper diagnosis; and this, in the majority of cases, would seem not such a difficult matter if the profession once realized the frequent occurrence of this disease and the fact that it is so often taken for acute rheumatism. It may also be said that prevention is often more possible and certainly more hopeful than cure.

PROGNOSIS.

This is usually rather gloomy. The general tendency of the disease is to progress, but we have to remember that in many cases the disease was never diagnosed until more or less permanent changes had occurred. How much can be done for early cases with early diagnosis we hardly have data to state. Reports have been obtained from 25 of the patients who had been in the hospital over three years ago. Of these only 2 could be described as well, 3 had died from an unknown cause, but only about half the remainder were much worse, 4 were crippled completely and 5 were only able to get about with crutches. The other half were much in the same condition.

There can be no doubt of the necessity of always giving a most guarded prognosis. They may recover, which is unlikely so far as our present knowledge goes, but we can generally only hope to keep the disease stationary by care and perseverance. It is to be hoped that early diagnosis may help the patients of the future.

"STILL'S DISEASE."

To this only brief mention will be given. There is some doubt as to the exact nature of these cases, but at present it seems best to include them among the cases of polyarticular arthritis deformans.

There were two cases in our present series, one a male aged 4, and one a female aged 5 years. They both had rickets, but there was no history of symptoms suggesting congenital syphilis. They both showed the symptom complex described by Still, namely, (1) an arthritis involving many joints, and with more change in the periarticular structures than in the joint itself; (2) an enlarged spleen, and (3) extreme enlargement of the lymph glands.

One patient was discharged somewhat improved, but the other went out completely well—one of the very few instances of recovery from this condition.

These cases seem to suggest an infective cause very decidedly. In the case with recovery the enlarged spleen and glands diminished with improvement in the general and articular condition. It seems most reasonable at present to consider them a type of arthritis deformans.

GROUP III. MONARTICULAR FORM.

These comprise a group of cases about which there is also much difference of opinion. The frequency is doubtful. Probably many of the cases often diagnosed as chronic rheumatism belong here, and also the group termed *Morbus coxæ senilis.* The disease usually occurs in elderly people, one of the larger joints, most often the shoulder or hip, is involved and the general type of change is degenerative. There is usually marked loss of function, wasting of the muscles about the joint, and sometimes ankylosis in the hip joint. In these patients very careful examination should always be made for slight changes in other joints. These may be very slight, but in some of them undoubtedly other affected joints may be found.

There are only 3 cases of the monarticular type in this series. The more chronic cases do not often seek hospital advice. The condition may not seriously interfere with their comfort, and they are content to make the best of it. Curiously enough, two of these cases suggest

in some ways an acute onset much like that described for the polyarticular form.

The symptoms may be noted briefly. Two of the patients were females, all were white, the ages were 39, 40 and 50 years. In two the shoulder was involved, and in one the hip. The former complained of pain in the hip and leg. In both the shoulder cases the onset had been rather sudden; they complained principally of pain and difficulty in moving the joint. Later the pain became less, but limitation of motion was left. The third case was more gradual. There was pain in the hip and down the leg, with gradually increasing stiffness.

The symptoms were much the same in all—pain on motion, distinct limitation of motion, crepitus, and some muscular wasting. In one of the shoulder cases the reflexes on the affected side were much exaggerated and normal on the other side. She also showed signs of slight involvement of two finger joints on the other hand. There were also some areas of pigmentation. The temperature in two averaged 99 F., in the third it went up to 100.5 F. The pulse rate in all was about 90.

The diagnosis in these cases may offer some difficulty. A gonorrheal arthritis can usually be excluded, and the greatest trouble generally is to distinguish it from a tuberculous joint. If the characters of the joint involvement do not give the diagnosis tuberculin may be used. We have found it very satisfactory. The finding of slight suggestive changes in other joints is conclusive.

Treatment is usually only palliative. Local measures, massage, wet packs, etc., are often of use, and in some instances surgical measures may give a more efficient joint, especially at the hip. But too often the degenerative changes advance gradually as in the polyarticular form. Every endeavor should be made to keep up the general health.

GROUP IV. SPONDYLITIS.

As already said, the cases with spinal involvement fall into two groups, one with general, the other with local

involvement. The former is readily diagnosed, the latter is certainly much more easily overlooked. These cases are usually considered to belong to the "osteoarthritic" group. The bony changes predominate, and especially new formation of bone. Certain writers hold that the osteoarthritic cases are quite distinct from the type previously spoken of as "rheumatoid" or those in which other than bony parts are especially involved. In certain of the cases of this series there seems no doubt of the *occurrence of both types in the same patient.* If this be true it goes far to prove that all the cases here grouped under the heading of arthritis deformans do belong to one disease which has various types. Not only were the two types of arthritis found in the one patient, but in some of those showing only osteoarthritic manifestations there was a history of attacks of acute polyarthritis exactly like those described under Group II, and from which the patient had recovered apparently completely. The histories are so characteristic that there seems little doubt of the nature of the old attacks.

There were 22 cases showing spinal involvement. In 13 of these there was spondylitis alone, while in 9 this was accompanied by symptoms in other joints; in other words, the lesions were of the polyarticular type, and these have been included in the analysis of Group II. In taking up the study of this group it is sometimes difficult to distinguish the symptoms from the spine and those from the other joints. In reference to the extent of spine involvement the cases were as follows:

	Gen. inv. of spine	Local inv. of sp.
Spine only involved[7]	6	7
Spine and other joints[7]	9	0

It is worthy of note that when the spinal involvement was associated with changes in other joints in no instance did we recognize involvement of a portion of the spine only. But, as said before, the confession is frankly made that only recently have we learned to

7. To prevent confusion it is convenient to refer to these two classes as the "spinal" and "mixed" cases.

diagnose the local form, and it is quite possible that instances of it have been overlooked. For the same reason the relative figures of these groups mean little as to their comparative frequency.

Incidence.—Of the 22 cases, 20 were males and 2 females. The striking preponderance of males is to be noted, especially as the other type is thought usually to occur much more frequently among females. All are white. The ages were as follows:

1 to 10	0	41 to 50	2
11 to 20	1	51 to 60	2
21 to 30	6	61 to 70	2
31 to 40	9		

Complaint.—This, in the majority, was of pain and stiffness in the back and legs. With this mention was often made of the crippling and general weakness. A few laid especial emphasis on the pain.

Onset.—The age of this was:

1 to 10	2	41 to 50	1
11 to 20	10	51 to 60	2
21 to 30	4	61 to 70	1
31 to 40	2		

Only 7 cases were admitted below the age of 30 years, but in 16 the onset was before 30. The onset in a few over the age of 50 is to be noted. The early age at onset in so many of the cases is striking.

The onset in the 13 spinal cases was gradual in 11 and sudden in 1, with one doubtful. In two of these there was a history of previous acute attacks of polyarthritis which subsided and left apparently no damage. These are exceedingly interesting. One may be noted briefly:

No. 61.—J. H., white, male, aged 45, was admitted complaining of stiffness of the back. There was a history of arthritis deformans in his family. From childhood he had at times some arthritis. With these attacks he was usually laid up, but never for very long. At 28 he had his first severe attack. This involved many joints and he was two months in bed. He recovered gradually and was well for seven years, when he had another attack with many joints involved, and the neck slightly. Three years and five years latter there were subsequent attacks, in the last one his spine becoming stiff. On examination the joint changes were practically confined to the spine and hips.

In other cases there seemed little doubt of previous attacks of local spondylitis, from which they recovered completely, and were practically well for many years. There were seven with very suggestive histories of these previous attacks with apparent recovery at any rate from symptoms. These intervening periods varied from two to eighteen years. One can not help wondering if in some of these cases it is only some accidental occurrence—such as an attack of influenza—which determined another attack. If this be true it may be that many cases may recover at any rate from ordinary symptoms of the disease.

General Features of the Spondylitis Cases.—These are most characteristic in the cases of general involvement. The spine is fixed and the term "poker-back" is most expressive. In some the head is held forward and there is marked bowing of the upper spine, and all rotation in the cervical region is lost. The patient to look about has to move the whole body. There is much difficulty in stooping, and on an attempt to pick an object from the floor they have to crouch down without any bending of the spine. On examination, the natural curves of the spine are often lost and the bowing of the upper dorsal and cervical regions is at times marked.

In the type with local involvement the picture is not so striking. The area involved is usually the lower dorsal and lumbar regions. The patient may make complaint of the back—commonly of pain and stiffness, which he may refer to the spine or dorsal muscles—or very often, of pain in the legs. This latter is common, especially over the course of the sciatic nerve, and there is little doubt that many of the cases regarded as sciatica are really arthritis deformans of the lower spine. Too often the diagnosis of neurasthenia is made, and perhaps also lumbago—whatever that term may mean. These cases on ordinary examination show nothing. It is only on the attempt to have them do certain movements that the

rigidity of the spine becomes evident. This can be demonstrated by having the patient stand with the feet together. The back should be visible. Then with the hips held, to prevent movement, the patient is asked to bend forward, backward and to the side. The restriction of free movement at once becomes evident, and it may be further shown by asking him to pick up an object from the floor. The marked interference with free movement between the vertebræ is very evident.

Certain curious features occur in some of the cases. There may be some alteration in the natural curves of the spine. Lateral curvature may be present. The upper part may be bowed with the convexity backward and the head held far forward. There may be marked prominence at the junction of the cervical and dorsal regions (1 case), or the lumbar curve may have disappeared (4 cases). There may be one prominent lumbar spine (1 case). The neck may only be partly ankylosed. Bony thickening may sometimes be felt about the neck vertebræ (2 cases). In 1 case the neck was rigidly fixed to one side. Occasionally the back muscles—especially in the lumbar region—may be very rigid or they may show wasting.

In some instances the hip joints are involved and there is restriction of movement there. In others, the symptoms are all referred to one leg, and the patient may systematically "save" it, and so usually walk on the toe or with the knee slightly flexed. Such cases are often mistaken for tuberculous hip disease. Some of these patients may limp very markedly.

Emaciation.—This is often marked, and was especially so in our group of mixed cases. With this there may be marked pallor, although this is usually more marked than the actual anemia. The local wasting is often very extensive. The muscles of the back, but especially those of the buttocks and legs, suffer. This does not seem to be always symmetrical. One side of

the spine at times may be more involved, and the wasting be greater also on that side.

Reflexes.—These are usually increased in the legs, both the knee jerks and the tendo Achillis reflex. With this increase in the legs, the arm reflexes in the local spinal cases are usually normal. In only one case was a diminished knee jerk noted. The cremasteric reflex was increased in 2 cases and an ankle clonus was obtained once. In 1 case with signs more on one side the reflexes, including the cremasteric, were all increased on this side and normal on the other.

Temperature.—Of the 13 spinal cases in 4 this was normal, in 5 it averaged 99 F., and in 2 it varied from 99 F. to 100 F. Of the mixed cases 3 had a normal temperature; these averaged 99 F.; 2 varied from 99 to 100 F.

Pulse.—In the spinal cases, 5 were normal (70 to 80 F.), 4 were between 80 and 90 F., and 3 between 90 and 100 F.

Blood.—In the spinal cases there are counts in 4 with an average hemoglobin percentage of 76; red cells, 5,205,000, and leucocytes, 7,800. The leucocyte count was over 10,000 in only one instance (11,300). In 4 mixed cases the averages were: Hemoglobin, 69 per cent.; red cells, 4,980,000, and leucocytes, 6,300. These findings are practically negative. The differential counts were normal also in both forms.

Tuberculin.—This was given in all the spinal cases and always with a negative result. It is a great help in the matter of diagnosis.

Radiographs.—These show a very characteristic condition. In all but probably the very early cases, definite deposits of new bone can be made out.

PATHOLOGY.

In this osteoarthritic group we have usually a marked overgrowth of bone. There is proliferation of the articular cartilage and later ossification of these outgrowths. Certain other structures may also be involved in the

bony change, such as the ligaments or fibrous tissue. These bony structures usually show little alteration in their histologic structure from ordinary bone. There is marked hypertrophy of bone in contradistinction to the type previously described where atrophy predominates.

In the spine the process frequently begins on the anterior aspects of the bodies of the vertebræ and extends along the ligaments. With this there is deposit of new bone. This may mean a more or less continuous bridge of bone, and in the radiographs of such cases the bony deposits can readily be seen between the bodies of the vertebræ. These may be on one or both sides, but in early stages are often unilateral. The subsequent course is very variable. The process may advance only slightly and there be comparatively little change. If the process be very active the intervertebral cartilages may be replaced by bone before any absorption occurs. In such cases we have the typical "poker-spine," simply one continuous bony column. Should there be gradual atrophy of the cartilages without replacement by bone, gradual curvature will result. This is seen very markedly at times in old men, who make little complaint of any symptoms. Should such a process occur more on one side we get a lateral curvature. All these various processes may occur throughout the whole spine or be limited to certain regions, most often the lower dorsal and lumbar.

These changes may also occur in the posterior portions of the vertebræ and the lateral or spinous processes be involved. The articulations may be involved both between the vertebræ themselves and with the ribs. This latter gives an interesting condition, namely, almost complete absence of any movement of the ribs and a practical loss of all costal breathing. This was very marked in some of the cases of this series. There may even be severe pain complained of—in the acute stages—when the patient tries to take a long breath, as was well instanced in one of our cases. Should the formation of

bone encroach on the foramina it is readily seen why pain referred to the distribution of the nerves passing through them is so common. This, too, may be on one side only, with unilateral involvement.

It is readily seen from the lesions why the symptoms are so varied. The stiffness is, of course, due to the local conditions. Pain may be in the back itself, but is very frequently referred to the areas of distribution of the nerves involved.

DIAGNOSIS.

As in the acute polyarticular form, the knowledge of the condition is an essential to its recognition. The cases of general involvement are so characteristic that a glance is usually enough to give the diagnosis. There is no need to dwell on them. But with the local forms the story is different. We have long been blind to what was before our eyes very frequently. To Dr. Goldthwait[8] of Boston much of the credit of drawing attention in this country to these conditions is due.

The symptoms complained of most commonly are stiffness, interference with motion, pain and muscular weakness. It may be that many of the indefinite pains complained of in the back, especially in the morning on waking, are due to this condition. Lumbago, while probably rheumatic or gouty in some, is perhaps at times due to this. Many of the cases of so-called "sciatica" are caused by arthritis of the spine, and also instances of obscure pains in the legs and about the body.

There is no intention of suggesting that every case of pain in the back or of sciatica is due to spondylitis, but it is suggested that in every such case it is most important to examine the back. Some cases otherwise regarded as neurasthenia with complaint of pain in the back (which is, unfortunately, only too common a combination), will be found to have local arthritis of the spine.

---

8. Boston Med. and Surg. Jour., vol. cxli, 1899, and vol. cxlvi, 1902.

Patients complaining of sciatica may have the same condition.

Of the methods of diagnosis first comes the examination of the spine. The patient should be completely stripped, or down to the hips at least. In the majority of cases little is to be made out. There may be some curvature, or a projecting spine, or more commonly an obliteration of the lumbar curve. There may be wasting of the dorsal muscles, or in the gluteal region, or of one or both legs. In some the attitude is suggestive. They stand with one leg a little flexed and "favor" that side. These results may be only suggestive. Next comes the investigation of the mobility of the spine. With the legs straight, the patient is asked to bend forward and touch the floor, bend back and to each side. Any limitation of motion is very readily recognized. This may be equally marked in all directions, or only in one, although usually at least two are combined. He may bend forward but a very short distance and to one side much less than the other, or lateral movement to one side only may be much restricted. These variations are very readily understood in view of the pathologic anatomy. These movements may cause pain, either local or referred. An attempt to bend to the right may cause the same pain in the leg of which the patient complains. The attitude on attempting to pick up an object on the floor is often characteristic. With this the sensation should be tested; areas of altered conditions are important. The reflexes are usually increased.

The diagnosis may be fairly clear from this examination, but there are two important aids, namely, tuberculin and the *x*-ray picture. The use of tuberculin excludes the most common source of difficulty, although tuberculous disease of the spine rarely gives the same disease picture as spondylitis. In some instances there is marked involvement of the hip joints with the spondylitis, and this may have led to a suspicion of tuberculous hip-joint disease. The radiograph, if it

shows anything, is usually characteristic. The deposits of bone usually appear as shadows between the bodies of the vertebræ. In early cases, of course, little may be seen, and in very fat patients it may be difficult to get a clear plate.

Should the diagnosis not be certain with any of these methods, one help remains—the therapeutic test. Put a light plaster jacket on the patient, which should extend from the axillæ to the level of the trochanter, and if there be spondylitis there should be marked improvement in the symptoms in a few days. This is especially useful in the cases with sciatica.

TREATMENT.

As in the other type, attention should be given to the general nutrition. The same rules for the care of the general health, fresh air, sunlight, nutritious diet and tonics (the syrup of the iodid of iron, arsenic, cod-liver oil, etc.), should be carried out. With these, massage, bathing, hot air and baking to a limited extent, may all be used. Locally, the great object to be attained is rest. This can not be done by keeping the patient in bed, unless the back be well supported, and this is difficult to accomplish properly. Some kind of jacket is more suitable. Perhaps the best plan is to put on a plaster jacket for two to four weeks, and then a strong enough leather one to serve the purpose. With the parts at rest, the pain lessens and the muscular irritability diminishes. In the cases with much curvature the muscular contraction is often largely responsible, and a month with the jacket may almost completely correct this. The jacket should be put on with the patient standing as straight as possible, but no force should be used. If there be deformity, the jacket will often gradually correct this, and a second one can be put on in two weeks. The jacket or support should be worn until the process is at a standstill, and later some lighter apparatus may be substituted. In such cases, after the condition is improved, every care should be taken to keep the

general health as good as possible to lessen the chances of subsequent attacks.

In the cases with general involvement the damage is done. Some form of light jacket may give relief, but the general health is the most important thing we can influence.

The early diagnosis is the most important consideration in treatment. But in the prognosis one has to keep in mind that the disease may advance steadily despite all treatment, and that the most that can be done is palliative.

CONCLUSIONS.

The points to which special attention is directed are:

1. The frequency of the occurrence of acute polyarthritis in arthritis deformans and the danger of mistaking this for acute rheumatism.

2. The importance of the recognition of the spinal forms of the disease, especially the local involvement.

3. That in arthritis deformans we have an obscure disease of considerable frequency worthy of study, especially from the etiologic aspect.

DISCUSSION.

Dr. James J. Walsh, New York—There is nothing more used to give lasting reputation to quacks than the treatment of chronic rheumatism. If the disease be examined very carefully it really resolves itself into a series of affections not rheumatic at all, nor does it have any relationship to articular rheumatism. The latter runs a definite course and is cured completely and no marks are left in the joints at all. Rheumatic arthritis considered as an acute disorder which leaves marks in the joints, is quite another affair. The term "rheumatism" is likely to cover many mistakes in diagnosis, and many errors and negligences. There are three things that are mentioned in the newspapers, catarrh, rheumatism and gout, and curiously enough the three words mean exactly the same thing; etymologically, they mean "to flow," or "to have excessive secretion." Few recognize the fact that acute rheumatism never becomes chronic rheumatism. Rheumatoid arthritis is another disease especially in this country, and is not affected by the remedies commonly used in the treatment of rheumatism. It is to be hoped that in the beginning of the twentieth century something will be done to clear up the

large group of cases called "chronic rheumatism" and bring a little order out of chaos. The word rheumatism has been abused to an enormous extent, and so long as practitioners are satisfied with this for diagnosis, rational therapeutics is impossible.

Dr. J. T. Moore, Galveston, Texas—During the past four or five years that I have been connected with the department of medicine in the University of Texas, I have been looking for just such cases as Dr. McCrae has reported to us. Two years prior to this I was demonstrating anatomy, and since then I have tried to keep up my connection with the department of anatomy. We have been astonished at the large number of cases we have had in the dissecting room with this disease, the changes occurring in the joints, and yet not recognized prior to death. Some of these cases were diagnosed as chronic articular rheumatism, and some were not diagnosed as rheumatism at all; yet we found various changes in the joints when they were examined in the dissecting room. As physicians we should enter more on the investigation of this disease. It is evident that most of these cases are not due to chronic rheumatism, although so often diagnosed as such, but to arthritis deformans. I know of one case in particular suffering from intense pains in the legs. I told the man that he had sciatica; then I afterward told him he probably had chronic rheumatism; but there is little doubt in my mind that if he were brought to the postmortem table some changes might be made out in the vertebræ, showing it to be arthritis deformans.

Dr. C. E. Skinner, New Haven, Conn.—The victims of arthritis deformans are numerous enough and miserable enough to justify a demand on their part that we put ourselves in a position to do something for their relief, and a correct and early diagnosis is the first and most important step in this direction. The majority of physicians confound this disease with others of an entirely different nature, prominent among which may be mentioned rheumatism, neuritis and gout. Rheumatism is comparatively easy to cure under modern therapeutic methods, neuritis is considerably more resistant than rheumatism, and arthritis deformans presents a very difficult problem under any conditions, and especially so when a correct diagnosis and the inauguration of a proper line of treatment have been delayed long enough for profound pathologic changes to have taken place in the joint structures. The condition of some of these victims of failure to recognize the ailment early is pitiable, indeed, from extensive ankylosis of the large joints, which condition complicates the therapeutic problem to a marked and sometimes totally unmanageable degree. The importance of correct and early diagnosis, moreover, is well brought out by Dr. McCrae when he says that arthritis

deformans is usually made worse by antirheumatic or antineuritic therapeusis. This has also been my experience. I know that it is almost, if not quite, impossible to differentiate some cases of arthritis deformans in the early stages from acute articular rheumatism, but in the majority familiarity with the clinical aspects of the subject and a proper degree of care and thoroughness in the study of the individual case will render possible a positive determination as to the nature of the pathology present. Then an appropriate line of treatment can be adopted at once and the patient will be saved from much future misery. More work should be devoted to the diagnosis of this disease, and when this is generally appreciated it will be better for our patients, and they will have less just cause for complaint.

Dr. Thomas McCrae, Baltimore—There are many aspects of the disease, and there were only two or three that I hoped to emphasize in my paper. I said nothing regarding the etiology; that is a doubtful matter, although the evidence, to my mind, points toward infection with one specific organism or several. The nervous symptoms may be explained by the fact that they are largely due to toxins.

With regard to early diagnosis, the importance of that is very great, and may give us the possibility of preventing later changes.

In regard to the treatment there are certain general principles which should be considered in both types of the disease. Good nutrition, sunlight, fresh air, and good food are essential. Patients who have not obtained those will be more likely to have subsequent attacks. The syrup of the iodid of iron is good, given alone or alternating with arsenic. This should be continued not for months but for years. Massage is usually helpful. Baths are of doubtful utility in many cases. When we consider the local treatment we should think of the two groups. In the osteo-arthritic form rest is very essential, but in the rheumatoid group exercise is essential. In the spinal cases the patients should be placed at rest for a time. In the rheumatoid cases if the patients are kept in bed there may be contractures, muscular atrophy and the joint changes may progress. In the treatment of such cases two things are very essential, viz., patience and perseverance, both in the patient and in the physician.

ALBUMINURIA IN DIABETES MELLITUS.

ITS IMPORTANCE AS A FACTOR IN THE CAUSATION OF DIABETIC COMA.

ARTHUR R. ELLIOTT, M.D.
Professor of Medicine, Post-Graduate Medical School
CHICAGO.

Among the secondary tissue degenerations which result from diabetes none is so frequent as lesional changes in the kidneys. No extensive autopsy reports are at hand to establish the relative frequency of renal changes in diabetes, but such as are forthcoming demonstrate in a striking manner the association. Thus out of Griesinger's 64 autopsies, renal alterations were found in 32. In the main, the changes were degenerative in character, either fatty or hyperplasic. Seegen found such changes in 20 out of 30 cases brought to autopsy, and Dickinson in 25 out of 27. No special form of renal defect in diabetes can as yet be looked on as typical, the changes being variable in kind and degree, although almost always present. The form which they assume will depend on the duration and intensity of the irritation, to which the renal gland structure is subjected. As the toxemia differs very greatly in character and degree in the different forms—mild and severe—of diabetes, so we find the secondary changes in the kidneys differing widely in their essence and microscopic character.

The most interesting histologic feature of the diabetic kidney is the hyaline degeneration of the tubular epithelium first described by Armanni, and now generally known as the cellular necrosis of Ebstein. This condition of the tubular epithelium is encountered only in

severe toxic forms of diabetes. Dreschfeld found these necrotic changes in 6 out of 10 cases of diabetic coma. Other renal alterations occasionally met with in severe forms of the disease are hypertrophy and the so-called lesion of Ehrlich, which, according to Frerichs and Ehrlich, consists in glycogen degeneration of Henle's loop and the straight tubes. All these renal changes may be ascribed to the action of toxic bodies of highly irritating character, such as acetone, diacetic and oxybutyric acids, etc., which are present in the blood and urine of such cases.

In the mild long-continued form of diabetes the renal changes which develop are usually those of chronic nephritis. The development of chronic nephritis under these circumstances may be explained by prolonged hyperfunction, combined with persistent but low-grade irritation of the renal structure by the saccharin urine, which lead first to hyperemia, and if long enough continued, finally to inflammation of the parenchyma and overgrowth of the interstitial tissue. In this type of the disease the renal gland changes are nutritional and degenerative, whereas in the more rapidly progressive and toxic form of the malady they are inflammatory or necrotic. In the majority of cases presenting chronic organic processes in the kidneys the nephritis is probably of the mixed type, presenting both parenchymatous changes and interstitial hyperplasia. In aged diabetes arteriosclerotic renal atrophy is frequent.

Whatever form the renal alterations may assume, they sooner or later give rise to the presence of albumin in the urine. The frequent association of albuminuria with diabetes has received considerable attention, and much statistical evidence is available.

Pollatchek	found	albumin in	37	per	cent.	of cases.
Von Schmitz	"	"	63.4	"	"	"
Lecorché.	"	"	50	"	"	"
Bouchardat.	"	"	45	"	"	"
Von Noorden	"	"	23.5	"	"	"
Grube	"	"	40.4	"	"	"
Stokvis	"	"	40	"	"	"
Bouchard	"	"	43	"	"	"
Von Dusch	"	"	28 5	"	"	"
Rokitansky.	"	"	66	"	"	"

These statistics represent an average of 43.68 per cent in which albuminuria was present in diabetes.

Attempts have been made to classify the albuminuria of diabetes. Goudard distinguishes two nosologic varieties, which he names functional and lesional, the former associated with renal fatigue with phosphaturia or with organic disassimilation, whereas the lesional is produced by some form of renal lesion. Jacobson, on similar grounds, separates albuminuria of diabetes into benign and severe.

Grube divides diabetic albuminuria into five varieties, viz.: 1, terminal albuminuria associated with diabetic coma; 2, cardiac albuminuria met with in connection with myocardial weakness; 3, senile albuminuria of arteriosclerotic origin; 4, functional albuminuria due to irritation of kidneys by the saccharin urine; 5, albuminuria associated with organic renal changes. Salles recommends an equally complicated classification.

It seems to the writer unnecessary and confusing to adopt any elaborate classification for this condition. With the exception of the rare cases of albuminuria produced by circulatory failure in patients of asthenic type, the albuminuria of diabetes may, for general clinical purposes, be placed in one of two categories.

I. TOXIC ALBUMINURIA.

To the class known as toxic albuminuria belong the cases of the severe form of diabetes which are entering the terminal stage of the disease and in which the highly irritating acid toxins of endogenous formation have produced hyaline changes of the tubular epithelium—the so-called cellular necrosis of Ebstein, already referred to. This albuminuria is never absent in the stage of organic disassimilation and in diabetic coma, although it may fail to make its appearance until the terminal stage is entered on and toxinuria becomes established. This albuminuria is never of severe grade, seldom

amounting to more than a mere trace, or at most, 1 to 1,000. It usually shows some increase just before death, and sooner or later becomes associated with large numbers of hyaline tube casts. After it makes its appearance, this form of albuminuria seldom disappears again completely. As a rule the albuminuria and glycosuria bear no direct relation to each other, and in occasional cases may even stand in inverse proportion to one another. It will usually be found, however, that a direct relationship exists between the toxinuria and the albuminuria, the latter increasing or diminishing with increase or dimunition of acetone and acid toxins in the urine.

This form of albuminuria constitutes what has been sometimes known as the acute albuminuria of McGuire. Universal clinical experience associates this form of albuminuria with the advent of coma, which gives it a grave prognostic significance.

II. DEGENERATIVE ALBUMINURIA.

The second class of albuminuria in diabetes includes the vast majority of cases of this condition, for notwithstanding the greater proportional frequency of the first variety to the class of cases in which it arises, the severe type of diabetes, these constitute but a small per cent. of the great bulk of diabetes. This form of albuminuria is also the product of renal irritation, but irritation of an entirely different nature and severity. In this class of cases the sugar in the urine is the offending agent. Its potency in this respect is much less than that possessed by the acid toxins of the severe grades of the disease, but its continuous action over long periods of time, combined with renal hyperfunction, which attends the associated polyuria, in the end inevitably initiate changes in the kidney which, in their full development, constitute nephritis. In proportion as the renal epithelium is affected, albumin appears in the urine, and as parenchymatous changes are usually a first result of the hyperemia following irritation, albuminuria is a

pretty regular attendant on the renal change. In many cases for considerable periods this albuminuria is not attended by any very demonstrable evidence of nephritis, and consequently it has been misnamed functional, to distinguish it from albuminuria, associated with definite and clinically recognizable renal lesions. It has been considered a development of little importance and in the presence of conflicting therapeutic indications, as between the glycosuria and albuminuria, the latter is usually neglected. After a time, however, this at first apparently accidental albuminuria is found to merge imperceptibly into a true lesional albuminuria, with impaired renal function and secondary cardiovascular developments. Hyperfunction and persistent irritation have done their work, and a nephritis of a mixed type becomes established and adds to the difficulties of the case.

It will be seen from the foregoing brief considerations regarding albuminuria in diabetes that fully a third or more of all cases of the disease manifest this symptom at some time during their course. Although by no means of itself always a sign of established renal mischief, it is never devoid of importance. It may appear so frequently without accompanying disturbance that many writers falsely regard it as a harmless sign. As pointed out, it may occasionally arise as the apparent product of irritation of the kidneys by the saccharin urine, but should the glycosuria continue, definite renal and secondary systemic changes eventually result. In aged diabetes with arteriosoclerosis albuminuria may be present from atrophic changes in the kidneys of sclerotic origin. In the late stages of diabetes, of whatever form, albumin may appear in the urine as the result of venous stasis from cardiac asthenia.

The conclusion forced on us by these considerations is that the development of this symptom is always a significant event, indicative in the mild, long-continued

form of the disease of developing nephritis, and in the severe toxemic stages of diabetes we are warranted by experience in regarding it as one of the terminal developments.

A matter of great interest in connection with the albuminuria of diabetes is the frequency with which the glycosuria is observed to diminish as the albuminuria becomes more pronounced. In exceptional cases the glycosuria may even entirely disappear, thus constituting the substitutive albuminuria of Salles. Maragliano is skeptical of a true substitutive process, and explains the fact on the theory that albuminuria appears on improvement of the diabetes under treatment, because the polyuria lessens *pari passu* with diminution of the glycosuria and albumin already present but masked by the previous dilution of the urine is revealed. This view can not be seriously accepted as an explanation, because in most cases diminution of the glycosuria does not precede the advent of albuminuria, but follows it, and albuminuria is, moreover, not usually the result of treatment. A diminution in the glycosuria, even its entire disappearance, has been observed to occur as the apparent result of the hyponutrition of such complications, as pulmonary tuberculosis, etc., when they arise during the course of a diabetes. This can not be considered a similar process to that under consideration, for in the great majority of substitutive albuminurias the nutrition is not materially depressed. Naunyn's contention that this peculiar conduct of the glycosuria is analogous to the behavior of that symptom in the presence of cachexia and marasmus, from whatever cause they may arise in diabetes, is stoutly opposed by Kleen, who observes that he has repeatedly seen cases in which nephritis, though advanced, has left a fairly good state of health, with no marasmus or cachexia, but with marked diminution of glycosuria. Such cases are within the experience of most observers.

A number of very suggestive experimental observations are at hand which have some bearing on this question of the diminution of the glycosuria in the presence of albuminuria.

Achard has suggested that in carrying out the test for alimentary glycosuria we must not forget that the failure of sugar to appear after large amounts of glucose have been swallowed, may be due not solely to the vigor of the sugar-converting power as has been supposed, but wholly or partly to renal impermeability. In confirmation of this idea he reports a case of apoplexy with a small amount of sugar in the urine but a very large amount in the blood (5.1 grams per liter). This hyperglycemia he attributes to a lack of renal permeability, which was shown to be present in this case by the methylene blue test. McGuire declares himself inclined to ascribe the diminution of the glycosuria to inefficiency of the kidneys, and he suggests that the albuminuria and its accompanying renal changes may be concerned in the production of coma.

Schupfer observed in cases of combined albuminuria and glycosuria experimentally produced that more sugar was excreted by the kidney which eliminated the least amount of albumin, the excretion of urea corresponding to that of sugar. When the kidney lesion was one-sided, the injured kidney excreted albumin and much less sugar than the uninjured one.

Ellinger and Seelig demonstrated that dogs from which the excretion of sugar was constant, after injection of cantharides showed a diminution in the output of sugar, the excretion falling in one case from 7.6 to 2 per cent. At the same time they were able to establish that the sugar content of the blood was markedly increased. They think that their results prove that the kidney lesion caused by the cantharides led to a diminution in the glycosuria, while the glycemia still existed. Along the same line as the foregoing are the experiments of Richter, who, after previously damaging the kidneys, pro-

duced glycosuria by means of phloridzin and observed that the excretion of sugar almost constantly began later than in control animals, and did not reach so high a point.

In an attempt to solve the question of the existence of a renal form of diabetes Klemperer administered as much as 10 grams of phloridzin to each of ten subjects having granular kidneys, and there was no reaction in seven. That three of his patients developed glycosuria he thought might be due to portions of the kidneys having been still unaffected by the morbid process. Klemperer gave phloridzin by the mouth. Pugnat and Ravilliod administered phloridzin subcutaneously to twenty-one cases of renal disease, and in no case was the excretion of sugar normal in amount, and they conclude that with renal disease phloridzin glycosuria is either diminished in amount or entirely absent. In thirteen of the cases a marked diminution was observed, and in eight cases it failed entirely to appear. The experiments of Achard and Delamarre, performed in the same manner as the above, gave somewhat similar results. In five cases of interstitial nephritis four showed a marked hypoglycosuria, and in one case no glycosuria resulted. In four cases of acute tubal nephritis three showed hypoglycosuria, and in one case no glycosuria resulted. Of nine cases of chronic tubal nephritis five were hypoglycosuric, and four non-glycosuric.

From these collected observations the inference seems clearly justifiable that increased tolerance for carbohydrates and diminution of glycosuria in the presence of albuminuria is due to lesion of the kidney, which operates to cause diminished permeability of the renal secreting structure, not alone to sugar, but also to urea and other extractives, with accumulation of these bodies in the blood.

In the light of this deduction, an entirely new importance becomes attached to albuminuria and its associated renal changes in diabetes. The development

of albuminuria in diabetes at once assumes a new and greater prognostic significance, especially in grave cases of the disease, where it may be of importance in the production of coma, by causing a heaping up of sugar and toxins in the blood. We have seen that there are constantly present in fatal cases of diabetic coma inflammatory and necrotic changes in the renal gland structure, comprising most frequently the epithelial necrosis of Ebstein, and less frequently hypertrophy with hyperemia and the degenerative lesion of Ehrlich. That these changes are not merely accidental is attested by the constancy of their occurrence, some form being almost invariably present. We have also seen that albuminuria invariably develops before the advent of coma. Grube, Williamson, Stokvis, Kleen and others regard its occurrence at this stage as constant, and their opinion is verified by practical experience. Personally, I have never seen coma develop without albumin being present in the urine, usually associated with casts. Kuelz first pointed out the frequent association of renal casts with diabetic coma, and they are now regarded as a sign of decided significance. We may regard the albuminuria and casts, under these circumstances, as the visible outward evidence of the renal gland changes. The results of experimentation already detailed point to the fact that the kidney lesions are inimical to the glycosuria, and frequently interfere completely with transudation of the sugar. Equal proof exists that urea, and presumably other organic extractives, suffer a diminution in the urine. It is a matter of common observation that immediately preceding the development of coma, a marked fall in renal efficiency takes place. Not only does the sugar diminish in quantity, but the quantity of urine, the urea and other urinary solids suffer a decided and material reduction. Dreschfeld has observed in two cases that at the outset of coma acetone and the ferric chlorid reaction entirely disappeared from the urine. No corroborative evidence is at hand regarding this interest-

ing observation. Certain it is, however, that preceding coma we invariably have renal conditions established with albuminuria and casts, and with decided reduction in renal permeability. To the writer abundant proof exists in these associated renal changes and clinical developments for the belief that the kidney alterations are an invariable antecedent of diabetic coma, and frequently its determining factor, by causing a sudden and marked diminution in renal permeability and a heaping up in the blood of the diabetic toxins. This view of the etiology of diabetic coma does not substitute a uremic for a purely diabetic origin, for the coma is certainly not uremic, although uremia may be a secondary contributing factor. It is frequently observed in grave diabetes that acetone and diacetic acid may be present for long periods of time without the incidence of coma, and not until the evidence of renal alterations make their appearance, does the patient develop profound evidence of coma. Experimental evidence regarding the toxicity of these bodies demonstrates that acetone, diacetic and oxybutyric acids can not be said to be toxic in the ordinary sense of the words, and that all of them can be taken by a healthy person in comparatively large amount without ill effects. It is shown, however, that if they are introduced into the blood of animals in large enough quantities they will produce symptoms closely resembling those of diabetic coma, and that they possess the power of producing albuminuria. It has been observed that acetone, diacetic and oxybutyric acids occurring in certain other states than diabetes occasionally give rise to all the graver symptoms of the coma of diabetes. It can not be doubted that these toxins are the cause of coma in diabetes, but it is the renal impermeability produced as a result of the irritation of these bodies in the blood and urine which is the final determining factor, by so increasing the toxemia through accumulation of these bodies in the blood as to initiate the coma:

CONCLUSIONS.

The following conclusions seem warrantable in the light of the facts here collected:

1. Albuminuria is a frequent development during the progress of diabetes, over one-third of all cases manifesting this symptom at some time during their course.

2. Albuminuria in diabetes may, for clinical purposes, be divided into two varieties, toxic and degenerative.

3. Toxic albuminuria is generally of acute onset, arising during the later stages of the severe forms of the disease, as the result of irritation of the tubular epithelium, producing hyaline degeneration thereof, and due to the presence of acid toxins in the blood and urine.

4. Toxic albuminuria is an invariable precedent and accompaniment of coma, and may be regarded as of the gravest prognostic significance.

5. Toxic albuminuria with its associated renal changes, is the final determining cause of coma diabeticum, by producing a heaping up of sugar and toxins in the blood, through diminished permeability of the renal secreting structure.

6. Degenerative albuminuria occurs with great frequency during the progress of the mild type of the disease, and is of renal origin and produced by a gradually developing nephritis, which arises from prolonged hyperfunction and impairment of renal nutrition.

7. The appearance of albumin under these circumstances, while of little immediate significance, is of the greatest eventual importance, as indicating the intrusion of chronic nephritis into the already manifold clinical difficulties.

8. A few cases of albuminuria in diabetes may be produced by venous stasis due to cardiac asthenia, and under such circumstances will be associated with other evidence of heart failure.

9. Albuminuria in diabetes is never devoid of importance. It may be of the gravest significance, as we have

seen, and is always of sufficient import to receive the earnest attention of the clinician.

BIBLIOGRAPHY.

Kleen: Diabetes Mellitus and Glycosuria, Blakiston's Sons & Co., 1900.
Von Noorden: Twentieth Cent. Pract., vol. ii, Wm. Wood & Co., 1895.
Kuis: Lyon Med., 1892, No. 23.
Williamson: Diabetes Mellitus, The Macmillan Co., 1898.
Grube, K.: Br. Med. Jour., 1898, No. 12.
Pollatschek: Zeit. f. Klin. Med., 1888; Sajous' Annual, 1888.
Schmitz: Berliner Klin. Wochen., April 13, 1890.
Klemperer: Le Sem. Med., 1896, p. 259.
Roberts: Urinary and Renal Diseases, Lea Bros., Philadelphia, 1885.
Goudard: La Presse Med., Dec. 15, 1897.
Saunby: Bradshawe Lecture, Lancet, 1890, vol. ii, p. 381.
Senator: Diabetes, Zeimssen's Cyclopedia, vol. xvi.
McGuire: Br. Med. Jour., vol. ii, 1886, p. 548.
Salles: Thèse de Lyon, 1894.
Jacobson: Manchester Medical Chronicle, October, 1896.
Maragliano: Gazz. degli Osped., March 5, 1890.
Schupfer: Il Policlinico, February, 1900.
Ellinger and Seelig: Verhandlungen des Congresses f. innere Med., Berlin, April 16-19, 1901.
Dreschfeld: Br. Med. Jour., 1886, vol. ii, p. 358.
Richter: Zeit. f. Klin. Med., Bd. xii., s. 160.
Achard and Castaigne: Le Sem. Med., 1897, p. 436.

DISCUSSION.

Dr. William Krauss, Memphis, Tenn.—The percentage of cases of diabetes with albuminuria is perhaps greater than stated by the essayist, at least this has been my limited experience. I recall a case that no longer eliminated sugar on account of lost permeability, only albumin and casts. He had a horror of Bright's disease, and so I assured him that he was suffering from secondary nephritis, and under proper regimen he did fairly well. A student one unfortunate day told him nephritis and Bright's disease were one and the same thing. This depressed him so that he took to his bed and never recovered.

Dr. F. R. Weber, Milwaukee, Wis.—There is one point of great importance in this connection, the great influence of intercurrent febrile affections in cases of diabetes. Symptoms of intercurrent febrile affections, lasting three or four days, have a wonderful influence on these patients. I recall an instance occurring in a young girl of eight years, who had diabetes in childhood, which was not a typical case at all. One evening the child was stricken with tonsillitis and died within twenty-four hours. In this case a physiologic albuminuria rapidly developed into a pathologic albuminuria, coma appearing before death.

Dr. Arthur R. Elliott, Chicago—My purpose in presenting the paper was to draw attention to the importance of albuminuria in diabetes; the association of the two having never been regarded hithertofore as having any special prognostic significance. I believe albuminuria occurring in diabetes is never without significance. In acute cases it ought to be interpreted as a sign of approaching danger, and it should under all circumstances receive the earnest attention of the practitioner. The association of albuminuria with developing diabetic coma is too invariable to be mere coincidence. The conclusions of this communication, if they be correct, make the development of albuminuria one of the most important incidents of the diabetic state.

THE PASSING OF CHRONIC RHEUMATISM.

JAMES J. WALSH, M.D., PH.D.
Lecturer on General Medicine at the N. Y. Polyclinic School for Graduates in Medicine.
NEW YORK.

In response to the question, "Have you ever suffered from rheumatism?" addressed to the ordinary patient over 40 years of age, the physician seldom gets a negative answer. Even younger patients will tell of having had vague pains in their arms or shoulders, or in their knees or ankles, that they consider to have been rheumatic. Popularly, the notion is that rheumatism is one of the ills to which flesh is almost inevitably heir, and, in the course of time, practically every one is supposed to have some touches of it. It is not too much to say, then, that rheumatism is popularly considered to be practically a universal disease. Physicians encouraged this idea, to a certain extent at least, by suggesting that vague pains are rheumatic in origin, or at least agreeing with patients when they say that probably they have rheumatism.

As a matter of fact, many of the irregular practitioners in recent years, particularly the osteopaths, draw their clientele (and it is by no means innumerous), from the ranks of the so-called chronic rheumatics. Most of the proprietary medicines, besides their other wonder-working effects, are advertised to be good for chronic rheumatism, and especially for that fateful condition, the uric acid diathesis, which is supposed to be provocative of chronic aches and pains of many kinds.

To talk, then, of the passing of chronic rheumatism in the sense of the gradual disappearance of the affection during recent years, would seem to be highly paradoxical. Never, more than at the present time, did patients insist on treatment for the relief of pains and aches supposed to be due to chronic rheumatic conditions of one kind or another. That there are many symptom complexes to which the name rheumatism is commonly applied is beyond doubt. How many of these pathologic processes have anything in their underlying basis of causation to justify the name rheumatism is a very doubtful question.

Chronic rheumatism would supposedly be a condition in which some of the pathologic conditions of acute rheumatism persisted, and in which, as a consequence of this persistence, tissue changes of various kinds took place, causing a deterioration of joint structures. As a matter of fact, however, it will be found that while very many patients confess to chronic rheumatism, comparatively few will admit having suffered from the acutely painful red swollen joint, accompanied by fever causing confinement to bed, which characterizes true acute rheumatism. It is now generally conceded that acute articular rheumatism is a specific infectious disease, and while other specific infections, as, for instance, gonorrhea, influenza or typhoid fever, may produce disorganizing conditions in joints, it is a typical sign of acute rheumatic arthritis to go on to complete recovery. When changes in joints remain after acute rheumatism there is always some suspicion of the possibility of secondary infection, or even a doubt as to the original diagnosis, acute articular rheumatism having been the proper one.

A little examination of the cases commonly called chronic rheumatism shows how many disparate conditions are jumbled together under this term. Vague pains of any kind, for which no direct cause can easily be found, are at once called chronic rheumatism. Many of these conditions can be traced to definite nervous and

mechanical conditions without much difficulty if careful examination of each individual case be made. I have already pointed out in previous papers that there are at least fourteen different achy conditions which are often called chronic rheumatism, but without any good reason. The most common form of pseudo-rheumatism is the relaxation of joints which occurs in the ankle and is known as flat-foot, or which follows the dislocations of joints producing tissue conditions which make the patients very sensitive for all their after-life to changes in the weather. Just as there are relaxations at the ankle joint, so there may be relaxations of other joints—the knee, the hip and the shoulder—which are apt to cause something of the muscular discomfort that becomes so prominent a symptom of flat-foot, or the use of dislocated joints in which relaxation has taken place.

A second very common form of vague pain usually attributed to rheumatism, occurs in connection with occupation neuroses. There is not a single occupation in which some movement involving the co-ordination of a number of muscles has to be made and frequently repeated, that may not be the source of pains and spasmodic inability to use muscles properly, such as occurs so commonly in connection with writer's cramp, or telegrapher's spasm. These movements need not necessarily be very complicated in order to have this discomfort develop. The lower limbs may be the subject of these occupation neuroses, as in the cases of men who work foot-power presses, or in those who run sewing machines, or even in nervous individuals who have to sweep much and constantly use the broom from one side supporting the weight on one leg, while the sweeping movement is accomplished with the arm. Filers, carpenters, laundresses who iron much, planers, men who have to use hammer and chisel, all suffer from these neurotic conditions, usually more painful on rainy days, and for this reason so commonly called rheumatism.

A third source of vague pains that may, if not care-

fully investigated, be attributed to rheumatism is neuritis. In persons who are exposed to the weather and who use one set of muscles much more than others, especially if at the same time there is the absorption into the system of such toxic substances as lead or alcohol, there not infrequently develops a neuritis of the nerve supplying the overworked group of muscles. Stonecutters, for instance, who use their deltoid and other shoulder girdle muscles for the purpose of frequently lifting a heavy hammer or mallet, and whose exposure to the elements leads them to take alcohol often in greater quantities than is good for them, develop a neuritis of that branch of the circumflex nerve which supplies these muscles. At first this makes the muscles much more easily tired than before. During this period other muscles in the neighborhood are asked to do more work than usual in order to make up for the paretic muscles. As a consequence, painful, overfatigued conditions develop. After a while, the neuritis continuing, there is almost complete loss of power in the deltoid, and then the man can no longer pursue his occupation.

It must be remembered that the branch of the circumflex nerve which supplies the deltoid muscle at the shoulder also sends certain filaments to the joint structures of the shoulder. These filaments are really trophic fibers, and as a consequence the neuritis set up in them causes a lowering of the nutrition of the joint structures. This, together with the forced disuse owing to the inability to use and co-ordinate the muscles of the shoulder girdle, leads to something resembling degenerative changes within the joints. Points of tenderness develop especially over the head of the humerus, and the absence of synovial fluid may lead to crackling and roughness, so that there is some grating or fremitus when passive movements of the joints are made. It is easy to attribute such conditions to chronic rheumatism and to consider the neuritis in the case as rheumatic in origin and perhaps secondary to the joint changes, if the examiner

does not follow out the history of the case carefully and examine what was the real beginning of the pathologic condition.

Some of these neuritic cases run a very insidious course, and it may not be clear for some time what the condition actually is. A very characteristic illustrative case of this kind has been recently under observation. The patient was a painter who did inside work, being occupied mainly with the painting of balustrades, doors, window-frames, and picture-moldings, requiring him frequently to occupy awkward positions and use many movements of the wrist in painting small carved portions of woodwork. He came complaining of vague pains in the forearm, and occasionally in the shoulder, which were worse on rainy days. He had been a painter for nearly ten years, and had never suffered from any symptoms of lead poisoning. There was no blue line on his gums. I concluded that it was a simple occupation neurosis, since the tired feelings that he described, and the tendency not to be able to work after a while, and the fact that the use of the brush in his left hand for a time had caused the development of the same condition, to a slight extent at least, in the other arm, were characteristic of such ailments.

One day he came to be treated for a sprained wrist. He had fallen on his right hand, and there was some effusion into the joints of the wrist. Two days later he appeared with a characteristic drop wrist. This had developed during the night, and his observation with regard to other painters rather led him to suspect what the true nature of the condition was. It is not unusual for drop wrist to develop after some injury to the part, just as it is quite a common story to have the first symptoms of toe drop as a result of alcoholic neuritis follow upon a sprain of the ankle. In both cases it is evident that for some time, probably for months, there has been a slow running, low grade and practically latent neuritis. It can easily be understood, however, how the

preliminary manifestations of this would be a fatigue of muscles coming on much easier than before the nervous affection began, and a tendency to neurotic conditions such as occur in occupation neuroses because of the difficulty of co-ordinating complex muscle movements when the nerve paths leading to the group of muscles are disturbed by the presence of inflammatory products.

A certain number of cases of so-called chronic rheumatism here in America which present evidences of degeneration in joint structures following acute attacks of arthritis, are undoubtedly not genuinely rheumatic in origin, but are of gouty nature. Gout is much more frequent in this country than has been considered to be the case. Ordinarily gout is considered an English disease, and we look for English heredity in those in whom we suspect its presence. The absence of such heredity is, in many physicians' minds, an argument against an affection being gouty. Ordinarily, it would be considered, I suppose, not too much to say that gout is at least four or five times more frequent in England than it is in America. As a matter of fact, the comparison of the statistics of patients admitted to Johns Hopkins Hospital in Baltimore suffering from gout with those admitted to St. Bartholomew's Hospital in London for the same affection are in the proportion of only three to four. That is, gout is three-fourths as frequent in this country as it is in England. How difficult it is at times to differentiate acute gout from acute rheumatism may be judged from the confessions of excellent clinical observers, including Professor Osler himself, that only after two or three attacks of supposed acute rheumatism have occurred in a series of years in patients under their observation did they find the gouty nodules in the ears or the tophi in the fingers which proved that former attacks had been gout, and not rheumatism.

Gout is, in this country, earned rather than inherited.

It occurs especially among those who are subject to the influence of lead and those who indulge too freely in malt liquors. While our beer is much less likely to cause the condition than the heavier malt liquors of England, there is now no doubt that the increased consumption of even light beer which has occurred in recent years in this country have made many more people liable to gouty attacks than before. As to lead poisoning, it must not be forgotten that besides painters and workers in lead factories, plumbers, typesetters, stereotypers and others engaged in the printing trades are likely to suffer from lead poisoning if they are susceptible to plumbum. The accumulation of lead in the tissues always predisposes to gout and leads eventually to changes in joints which may be described as chronic rheumatism if the real nature of the pathologic condition and its true etiology are not recognized by careful elicitation of the history of the case.

In recent years the condition known as rheumatic gout, or rheumatoid arthritis, or, better than any of these names, since it does not assume a knowledge of the etiology of the condition of which we have not yet described well, arthritis deformans, has occupied much more attention. We now know that this condition may occur in three forms. There is the slow running form, which begins usually as Heberden's nodes, and sometimes, though not always, goes on to involve other joints besides the terminal joints of the fingers, and so causes deforming, disabling deformity. The pathologic basis of this is a true productive osteitis of the ends of the bones near the joint surfaces, which seems to be due to the presence of irritants in the blood. It occurs by preference in nervous people. The acute attack of pain and discomfort in the affected joints, which are noted especially at the beginning of the affection, are likely to be more frequent and more annoying whenever the patient is under emotional stress or suffering from worry for any reason.

The second form of rheumatoid arthritis is usually called osteoarthritis and in contradistinction to the first it occurs more commonly in men than in women; it is seen by far most frequently in elderly male subjects. It consists of a true osteoarthritis usually confined to the hip joint and spoken of by orthopedists not infrequently as senile coxitis. It may occur also in the joints of the spinal column, producing a certain amount of soldering together of the vertebræ and consequent fixation of the back, which gives a very characteristic gait. Like the affection of a single large joint, this spondylitis deformans may be absolutely confined, as its name indicates, to the vertebral column, and is, in fact, usually so confined. This form of the arthritis deformans may occur in younger subjects than the coxitis deformans already referred to.

Neither of the two forms of rheumatoid arthritis described are likely to be taken for rheumatism by a carefully observant practitioner, though very few patients suffering with these affections come under treatment without being convinced that they are suffering from an affection of rheumatic origin. The third form of rheumatoid arthritis, however, has many symptoms in common with rheumatism, and is undoubtedly one of the main reasons why acute rheumatism is thought to produce chronic pathologic conditions in joints oftener than it really does. This third form of arthritis deformans, called by English observers acute rheumatoid arthritis, is described very well by W. Hale White in the last number of Guy's Hospital Report, Vol, 57, 1902.

This third form of arthritis deformans occurs for the most part in women whose age is usually not far from 20. The disease begins, or is at any rate most prominent, in the proximal phalangeal joints, differing in this from the slow-running form of arthritis deformans which in elderly women begins in the distal phalangeal joints. Acute rheumatoid arthritis is usually strikingly seen in the wrists. It is markedly symmetrical, as a

rule, in both hands. The swelling is considerable, and extends beyond the affected joints, so that there is a distinct fusiform swelling about the interphalangeal articulations and a general swelling about the wrist and other joints. Sometimes a creaking may be felt, due apparently to thickening of the synovial membranes. In the course of a very few weeks, as a rule, most of the joints in the body are affected, so that the patient becomes bedfast, and is unable to move fingers, thumbs or wrists, and sometimes not the elbows, shoulders nor even any of the joints of the lower extremities. The joints of the spine are often affected, so that turning in bed is difficult and painful, and the temporomaxillary joint is frequently implicated, so that mastication becomes a matter of great discomfort.

Muscular wasting is so very rapid that it is clearly an extreme degree of genuine arthritic atrophy not dependent on disuse. The wasting is usually seen best in the hand and forearm, but in a bad case all the muscles may become distinctly atrophic. The most interesting thing about the disease is the temperature. There is a daily rise toward evening up to 101 F. or 102 F. Exposed parts of the patient are likely to be covered with sweat, and, as can be readily understood, there is strong simulation of acute articular rheumatism. The points of differentiation from articular rheumatism are quite clear, however. The disease begins in the fingers, gradually affects all the other joints, seldom fails to involve the temporomaxillary joint, and very commonly affects the joints of the spine, which are seldom attacked by true acute rheumatism. The disease runs a definite course, the temperature coming to the normal after three to four weeks. Much immobility and some swelling around the joints remain. Most patients have a distinct tendency to relapses during which the joints are once more swollen and tender as before. After the febrile stage is passed swelling of the synovial membranes and the soft tissues around the joints remains and the muscular atrophy

causes disability. This condition may not improve much, and the patient may remain a chronic invalid.

It is easy to understand how cases of this kind, which have been described by such excellent observers as Garrod and Bannatyne, as well as Hale White in England, would give the impression of an acute rheumatism running over into chronic rheumatism. This form of arthritis deformans is, however, a disease by itself, usually following some infectious fever, as influenza, parotitis, tonsillitis or the like, and perhaps itself of specific microbic origin.

It is easy to understand, from all that has been said, what little space is left for so-called chronic rheumatism. There would seem to be a few cases of true acute rheumatic arthritis, or of the subacute variety which produce changes in the joint tissues that are persistent and progressive. There is a growing impression that even of these few cases the majority are due not to rheumatism alone, but a mixed infection, the secondary infection being responsible for the persistent pathologic lesions. Certainly cases of true chronic rheumatism, that is of progressive pathologic changes in joint tissues for which no other cause except rheumatism can be found, are very rare. So much so, very probably, as to constitute them a medical curiosity. Personally, I should like very much to see such a case, for I am free to confess that I have not as yet seen one.

Is it possible, then, that chronic rheumatism is to be discussed without even a mention of the uric acid diathesis. Personally, I see no reason for hauling in a diathesis for which we have no substantial proof. Certainly the vague pains that are usually attributed to the uric acid diathesis or to lithemia are, in my experience, due more to a neurotic constitution as a predisposing factor and overwork of particular groups of muscles usually under unfavorable conditions as a direct etiologic agent, than to any other cause.

This, of course, does not deny the existence of any

of these achy, painful conditions, which are so common and so frequently demanding treatment. It does not deny that during damp weather, and in people who work or live in damp locations, there will be a distinct tendency to chronic aches and pains, just as there would be to frequent toothache under the same circumstances. It only transfers these affections from the column of chronic rheumatism, where they are all jumbled together, to the manifest opprobrium of our therapeutics, to separate columns, with regard to some of which we know our therapeutic impotency, but with regard to most of which we are fully aware that massage, rest, local treatment and improvement of the constitutional condition will do much to get rid of the symptoms. If the general public and the majority of the medical profession were once persuaded that chronic rheumatism, instead of being a common ailment, is, indeed, very rare, then there would be less failures of diagnosis, with consequent inevitable failures in treatment and less resort to our osteopathic friends, the enemy, and to the many remedies that are supposed to alter the uric acid diathesis and get rid of chronic rheumatic tendencies.

DISCUSSION.

Dr. James Tyson, Philadelphia—Dr. Walsh has essayed a difficult subject, difficult as are all subjects characterized by an unknown or at least inexact pathology. There is confusion between gout and chronic or subacute rheumatism. While I think it is true that gout is a more frequent occurrence in America than has been supposed, I do feel sure that a great many cases are called gout without sufficient reason. I do not think a diagnosis of gout is justified unless there is or has been podagra, or uratic deposits in the joints or evident hereditary tendency. Heberden's nodosities, regarded as gouty by so many, may be gouty, but more frequently perhaps are a product of rheumatoid arthritis. I wish to urge on my fellow practitioners of medicine an attempt at more precise diagnosis of the two conditions. I am sure that in certain cases in my own experience many years have been required to determine whether a case be one of gout or rheumatism.

I should like to say a word also on the threadbare but still undetermined subject of the uric acid diathesis. I sympathize with Dr. Walsh in this respect, and it is mortifying

to note how often rheumatic pains are ascribed to the uric acid diathesis without any reason whatever. It is often a careless expression, and not always intended by those who use it, but it gives an impression to the laity which is erroneous. As Dr. Walsh says, we really do not know what the uric acid diathesis is. There is, however, a clinical complex consisting in a scanty secretion of urine, of high specific gravity, with constant and copious deposits of uric acid and urates which is associated with the uric acid diathesis and is evidence of its presence. But it must be a permanent condition. It has doubtless happened to many of us to note in the morning after a liberal dinner including wines a scanty urine with uric acid deposits, etc., which will wear away after liberal ingestion of water, but this temporary state is not the uric acid diathesis. In the conditions referred to even the ingestion of large quantities of water does not increase the amount of urine secreted, and the tendency to sediments continues. To this condition I think we are justified in applying the name "uric acid diathesis." Such cases are not very numerous, and it may happen that associated muscular and joint pains are the result of it, but one ought at least to be sure of the combination before one asserts the causal relation.

Dr. Thomas McCrae, Baltimore—Dr. Walsh has faced a question that the majority of us have faced frequently, and often with the diagnosis of "chronic rheumatism." The profession is largely to blame for the ideas the public have regarding this condition; we inherited the idea and pass it on. Dr. Walsh brought out a good point when he said that if one made a diagnosis of acute articular rheumatism and, after the attack is over, joint changes persist, then consider a wrong diagnosis; if we did that we would find fewer mistakes.

Dr. Alexander Lambert, New York—This helter-skelter diagnosis of rheumatism has been made for a long time, and my confrères in New York should take a more self-respecting attitude toward these painful affections. Often the diagnosis is easily made. It has for a long time struck me that so-called cases of chronic rheumatism with active tissue changes, ulnar deformities, etc., have nothing to do with rheumatism. It is perfectly true, in my opinion, that many cases began like articular rheumatism which can not be differentiated from it, which later prove to be true cases of arthritis deformans. In my experience this is seen most commonly in young women.

Gout is much more common in New York of late years, and it is certainly more often met with in hospital work than formerly. A great many cases that at first seem to be cases of rheumatism and yet which are treated as gout get well, and this is especially so in women where you get so often this irregular type of gout. That type of real chronic rheumatism

is, as Dr. Walsh has stated, one that we must disregard. There are cases of rheumatism which begin as acute articular rheumatism, and before the patient recovers from one attack entirely they may have another acute attack and then run on into a subacute condition that one must look on as chronic, a chronic articular rheumatism. This process runs along a long while and leaves the patients more or less crippled. A great number of mistakes come from calling real gout rheumatism, and the ideas that are so prevalent regarding the uric acid diathesis have much to do with it. I am only too glad to be able to add my protest against this asylum for lazy diagnoses. I think it is firmly believed that the uric acid diathesis, occurring in healthy, vigorous, athletic, strong young men, is the cause of nearly all the woes they are heir to, especially the muscular pains. Whatever causes gout causes uric acid, and uric acid is simply one of the many symptoms. These men were usually 20 years old, and no one would accuse them of being gouty or rheumatic; they were in their prime and in vigorous health, and yet, among such, uric acid deposits in the urine are frequently found.

I wish to thank Dr. Walsh for bringing this subject up, because it is only by such papers and by such discussions can we realize that the so-called cases of gonorrheal rheumatism, scarlatinal rheumatism, etc., are not cases of rheumatism at all, but distinct and definite diseases; all cases naving pains in the joints should not be always considered as rheumatic, but they should be clearly differentiated. It is only by so doing that we will be able to be honest with ourselves and arrive at an accurate and scientific knowledge of the subject.

Dr. James J. Walsh, New York—When I called my paper "The Passing of Chronic Rheumatism" I did not mean absolutely no cases of chronic rheumatism existed; I did mean to say that cases of chronic rheumatism were more or less curiosities and well worth studying. Chronic rheumatism is generally thought to be very common. If one should run across a case of true chronic rheumatism we would want a report of it in our journals. What is acute rheumatism? We think it is an acute infectious disease. A number of clinicians think the disease runs a course of from two to six weeks and gets better; they give salicylates, not because it is a specific for rheumatism, but because it lessens the pain and lowers the temperature. Again we have certain joint lesions occurring in gonorrhea, scarlet fever, typhoid fever and intestinal troubles that are accompanied by diarrheas, dysentery, etc., all of which may be accompanied by joint lesions and to which the term "rheumatism" is applied. I wish to enter a plea for a better knowledge of this condition.

It gives me pleasure to hear what Dr. Tyson has said regarding the uric acid diathesis; I think the uric acid diathesis has

been overdone when we say that it gives rise to so many aches and pains, etc. If one looks into the literature one will find little authority for thinking the uric acid diathesis a prominent factor in disturbances of metabolism. We must remember that nervous people are more subject to pain than others; they may simply have some discomfort and think they really have pain, and such is particularly worse on rainy days. Dampness is associated with it.

HEMOGLOBINURIC FEVER.

ITS CAUSES AND TREATMENT, WITH ESPECIAL REFERENCE TO THE USE OF QUINIA.

WALTER SHROPSHIRE, M.D.
YOAKUM, TEXAS.

In presenting this subject for your consideration, we do so because of the diversity of opinion concerning it, hence evident need for more thorough study and discussion. Our consideration must leave out many of the features and characteristics of that arch mimic of all diseases, malaria, and be confined to that disease characterized by hemoglobin in the urine and due to malaria in some of her protean manifestations, discarding for the time Yersin's supposed discovery of a specific cause other than the malarial plasmodium, and all other theories and suppositions born of efforts to account for this peculiar manifestation.

In making an effort to clear up some of the uncertainty enveloping the subject, and to arrive at the truth concerning this disease, it was determined to depend, not alone on the individual conclusions of those who saw the disease, influenced no doubt by former teachings and impressions, but to get as thorough report of cases as possible, and view them, their cause, treatment and results, singly and collectively, drawing conclusions from recorded facts, and not by one individual or from one point of view, but by men of many localities, teachings and impressions.

Accordingly 2000 blanks for report of cases were printed, which provided for a thorough report of all features that we are to consider here, together with some

others that are desired for future consideration, and with a letter requesting a report of cases were mailed to the physicians of southern and eastern Texas and Louisiana, selecting localities where it was presumed the disease most prevailed. Of those requested, 81 replied; 40 of whom could not report cases, while 41 reported 173 cases, to which I added 29 cases of my own, making a total of 202 cases reported by 42 observers. Many of those reporting had treated many times the number of cases reported, but owing to the lamentable fact that no case book was kept, they were unable to make even a partial report on others.

Of those cases reported many features of the disease were unreported on, owing to the fact that in most instances the patient was in the rural districts, so far from the attendant's office that frequent and proper observations could not be made; and when the patient was seen many of the accoutrements necessary for proper observation were not at hand. Therefore, in tabulating and reporting on the different features observed, it will not be of the whole 202 cases, but of that number on which these features were reported.

Of the 202 cases collected there were 61 cases treated without methylene blue or with less quinia than 15 grs. per diem, which is practically no quinia, of which 26.2 per cent. died and 73.8 per cent. recovered; while 112 were treated with 20 grs. or over of quinia, of which 16.9 per cent. died and 85.1 per cent. recovered; 4 were treated with methylene blue, with 1 death and 3 recoveries.

Of the cases treated without methylene blue or quinia 30 per cent. had hemorrhage from other organs than the kidneys, occurring on the second, third and fourth days, evidencing a non-subdued condition of the malaria and its recurrence, involving other organs; of these 50 per cent. died, two during the chill following the hemoglobinuric attack, and one during the second chill thereafter.

Of those treated with quinia, 9.8 per cent. were so affected, with 3.6 per cent. fatality. It thus appears that quinia effects not only a lower death rate, but also lessens the percentage of recurrent paroxysms. In those cases treated with quinia your attention is invited to the following facts which bear on the adequacy of the dosage: Of 50 cases treated with 20 to 30 grs. per os, 22 per cent. died; while of those given 30 grs. or over in the stomach, 16 per cent of the 43 died; and of those given 40 grs. or over only 8.6 per cent. of the 35 died. Of the 19 cases treated with quinia hypodermically, only one case or 5.26 per cent. died, and that one within 48 hours of the onset, of toxemia, due to the paroxysm had before treatment was begun. There were 12 cases reported where the urine became darker upon the administration of quinia and three where it became darker when quinia was withdrawn. Of the total of the non-quininized patients, 9.8 per cent. died of uremia, while of the 19 deaths among quinia patients, 8.9 per cent succumbed to uremia. While uremia occurred in 16 per cent. of those treated with less than 30 grs. by the stomach, it occurred in only 2.8 per cent. of those treated with 40 grs. or over by the stomach, and not at all in those treated with quinia hypodermically. The average illness of those treated without quinia was 9.66 days and of those treated with quinia, 5 days. Of the men reporting on the point, 29.41 per cent. believe quinia may and does produce hemoglobinuria, while 70.59 per cent. believe that it does not. The largest dose of quinia that seemed to produce hemoglobinuria was 40 grs., while the largest which did not produce it was 100 grs. The average largest dose that seemed to produce hemoglobinuria was 13 grs., while the average largest dose that did not produce it was 52.6 grs. The smallest dose that seemed to produce hemoglobinuria was 4 grs. and the average 6.4 grs., while about 15 per cent. of cases occurred when no quinia had been given for 24 hours or over. The total average dose that eeemed to produce hemoglobinuria was 9.7 grs. per day.

In each case here estimated, the subject had been living in a violently malarious locality three months or longer, and most of them for years. In all save one there was history of malarial paroxysms immediately preceding the pernicious or black water form, and in all a previous malarial development. The number of men reporting blood examinations was 4. Number of cases, 12; parasites found in 5 cases, not found in 7.

The following conclusions are justified:

1. This disease always occurs in persons suffering repeated attacks of malaria (this was so in every case reported).

2. It nearly always follows one or more mild paroxysms of malaria at the proper time for its next exacerbation (there was but one exception to this reported).

3. It has all the characteristics of malaria, chill, fever and sweat.

4. Where adequate examination of blood is made, the hematozoa of malaria are found (it was so in 41 per cent.).

5. Its habitat, that of most violently malarious districts (all cases reported were so) are sufficient to establish malaria as a causative agent, if not the only one.

To the present there is no claim for a cause other than malaria worth more than a passing notice, except the one which is maintained by about 30 per cent. of those reporting, namely, that quinia produces it, to which claim we invite your careful and scrutinizing attention.

To establish the cause of any disease we must apply the agent to the subject and have, as uniform result, the disease. But if there are two agents suspected as causative which applied together produce the disease, but applied separately to the same individual the one produces it, the other never, we can attribute only to the one a causative place and to the other an accidental presence.

Such is the case before us. Malaria, taken as a cause and applied without quinia to an individual of such ten-

dency, hemoglobinuria results in 15 per cent. of the cases before us. Quinia had been applied to probably all of the cases before us without the presence of malaria and no hemoglobin appeared in the urine. Then in 85 per cent. of them malaria and quinia were both applied to the individuals, hemoglobinuria resulting. Which produces it?

Physiologically quinia acts on the blood, nerves and small vessels. On the blood it inhibits the oxygen-carrying power without causing separation of the hemoglobin from the corpuscle. On the nerves it is, first, a stimulant, then as the dose increases a depressant till it suspends the function of some of them entirely, as the optic and auditory, and in lethal doses, those governing the heart.

On the blood vessels it acts as a vasoconstrictor, increasing blood pressure and toning up those areas where from malarial toxemia there is a stasis, as in the brain in some forms, and the gastro-intestinal tract in others, and in the case before us, in the kidneys. And, like all such remedies, when increased sufficiently it ceases to act as a stimulant to the vasoconstrictor muscles and acts as a paralyzant of them, hence a dilator of the vessels, this occurring just as the lethal dose is reached, which, added to the suspending of the function of the vagus, causes such lowered blood pressure that death soon ensues.

Reviewing these effects of quinia it is indeed difficult, if not impossible, to conclude logically that it can produce hemoglobinuria. To consider it from an experimental standpoint, we observe that the deaths produced by quinia have been due to its effects on the nervous and circulatory systems, with never an occurrence of hemoglobinuria. If quinia is capable of producing this condition at all, it looks strange that lethal doses have never produced it, while those who believe it capable of doing so attribute the production to an average daily dose of 9.7 grs. Thirty of the cases before us had the disease without the exhibition of quinia and of those who were

taking quinia when the hemoglobinuria developed, in 89 of the 108 under increased dosage it disappeared. Indeed, it appears to me that one must be a fit subject for homeopathy to conclude that quinia, having produced the disease in 108 cases, would cure it in increased dosage in 89 of them. Again, in those where quinin might be supposed to produce the disease it were untenable logic to maintain that removing the cause would result in an increased death rate, while increasing the cause would diminish it, as 26.2 per cent. is to 16.9 per cent.

Of those reporting, the maximum dose of quinia given without producing hemoglobinuria, the largest dose was 100 grs., and the average 52.6 grs., and of those reporting the largest and the smallest dose that seemed to cause hemoglobinuria, the largest dose was 40 grs. and the smallest 4 grs., or an average of 9.7 grs. per day. Now, in the face of reason, can we maintain that 9.7 grs. of quinia will produce the condition when 52.6 grs. fail to produce it?

Those cases where quinia has seemed to produce hemoglobinuria have invariably been in persons known to be malarially infected, where the disease seemed mild and quinia was administered to eradicate it. Carefully considering these cases, we will note that of 1000 cases so treated probably in 950 cases the disease disappears, one develops the hemoglobinuric form, one the comatose form, three the enteric form, while 45 will return still in the mild form. There is unquestionably as great a percentage that develops the comatose form, due to peculiar determination of the disease to the brain, and three times as great a per cent. that develops the enteric form, due to the determination of the disease to the intestinal tract, as there is of the hemoglobinuric form. Now, if the quinia produced the hemoglobinuric type, did it not also produce the comatose and enteric forms? And how about the 45 returns of the mild form? Did quinia produce them also? Is it not as rational to attribute the

one to quinia as the other? Or is it not a fact that in all of the cases which recurred, the remedy had failed to effect the cure and the disease continued, in some assuming a more pernicious form, in others continuing the mild type? It appears that such is the case and that its assuming certain forms as it grows severer or its selections of certain organs on which to exercise its severest toxic effect, is one of individuality or peculiarity of the subject and not of the remedy. If quinia is to be eschewed in treating the one form, hemoglobinuric, should it not be shunned in the others, comatose and enteric? Yet I dare say that no man in this assembly would have the temerity to withhold quinia, the only specific, in the comatose or other pernicious forms of malaria.

Another strong evidence that it is the individual peculiarity that determines the hemoglobinuric form, is the fact that this form recurs again and again in the same individual and is prone to run in families, while seldom indeed an individual who has suffered an attack of hemoglobinuria develops a pernicious malarial attack other than the hemoglobinuric form. Why is it thus, if not due to the individual peculiarity?

Another error in the conclusion that quinia produces hemoglobinuria is the fact that in the vast majority of those cases quinia is withheld during the greatest pyrexia of the preceding attack, or to wait the action of calomel, and is administered too late to prevent the pending paroxysm; this growing more severe assumes the hemoglobinuric form, or, if it has already become hemoglobinuric, the urine that has partially cleared during the interval becomes darker, which is attributed to quinia. In this connection permit me to say that in every case seen where quinia had been administered and might be supposed to play a part in producing hemoglobinuria, the paroxysm occurred at the regular time for the exacerbation of fever and with chill and rise of temperature, just as occurs when the disease is left to

malaria alone; and in those cases reported by others, even where black water occurred as often as three times, the increased color of the urine marked well the time for recurrence of malarial paroxysm in approximately 20 per cent. occurring on alternate days, 75 per cent. on successive days and 5 per cent. twice daily, just as we find malaria acting in other forms than this.

Bearing in mind the established fact that the malarial parasite is most easily killed by quinia in its younger existence, it is easy to understand that withholding the drug through the stage of high fever or till the effect of calomel is had, will permit the growth of the plasmodium to such a size as to be decidedly more resistant to quinia, and to allow it to go on to sporulation, thus producing another paroxysm which may be pernicious, even hemoglobinuric, and still there be enough quinia in the blood to kill the young parasite as it is set free in the plasma, thus explaining some of the spontaneous cures without further administration of the drug.

Another error in quinin therapy of this disease, to which your especial attention is invited, is inadequate dosing. The estivoautumnal parasite, the one most commonly producing this disease, is far more resistant to quinia than other forms, and it, like all forms of animal life, becomes habituated to any poison till it can withstand even ten times the dose necessary to kill when not so habituated. In all violently malarious districts, such as this disease prevails in, and especially in every chronic malarial subject, there is, without exception, in this country, a partial saturation of quinia for a considerable time, if not by quinia prescribed as such, then by quinia in the form of the numerous patent chill tonics, which have for their base quinia; hence the malarial plasmodium in this disease has, to some extent, become a quinin habitué, and only vastly increased doses prove toxic. Personally this has been observed many times; in one instance,

25 grs., given hypodermically to a year old child suffering from the enteric form, although administered 8 to 20 hours prior to the pending paroxysm, failed to prevent it and the little fellow died during the chill. You will observe that only 8.6 per cent. died where the dose was 40 grs. and over, while 22 per cent. died with 30 grs. or less.

Your attention is called to another point of error in quinin therapy in other forms of malaria as well as this, and it is by far the most important point of all—that of failing to get the drug into the blood. It is adequately proven and generally conceded that the malarial parasite destroys its host, the red blood cell, on sporulation, thereby setting free into the plasma what hemoglobin it has not consumed, there to remain until the liver has converted it into bilirubin. This, frequently repeated and in excessive quantities in the pernicious types, overwhelms the liver with work till its function is almost, if not quite, suspended, and a stagnant portal circulation results, hence the *vis-a-fronte* causes a passive congestion of the stomach mucous membrane and a cessation of its functions, both secretory as to hydrochloric acid and absorptive. The first of these conditions evidenced by alkaline vomitus, would prevent the solution and absorption of quinia put in the stomach in other than an acid solution, were we able to keep it there, while the abolition of the absorptive function would prevent the taking of quinia into the blood were we able to maintain an acid solution on the stomach; or more accurately speaking, the blocked portal circulation would prevent its entrance to the general circulation and its application to the parasite. So quinin dosage by the stomach is in many instances but a method of deceiving ourselves in the belief that we have applied this specific in a case where it is needed, while in fact the drug has never reached the blood where the parasite lives.

Just here your attention is called to the fact that all the cases where quinia was thought to be the cause of

hemoglobinuria have been cases where it was given by the stomach and by men who depend on the stomach administration, while not a case receiving hypodermic administration developed the disease and none of the men who depend on the hypodermic method attribute the disease to quinia; the death rate under the hypodermic method being 5.2 per cent., while that under stomach administration is 20 per cent. This, taken in connection with doubtful ability to saturate the blood through the stomach in pernicious forms, seems quite significant.

It is claimed that hemoglobinuria is the manifestation of an idiosyncrasy for quinia. Yet no case of unquestioned freedom from malaria has been observed to develop hemoglobinuria from quinin administration, and in every case observed and reported the patient had taken quinia for other troubles and mild forms of malaria without the development of hemoglobinuria. If quinia produces it in severely malarial subjects, why not in mild and non-malarial ones?

Those cases attributed to quinia are not experimentally produced or based on physiologic or pathologic facts, but on purely paracelsian empiricism. Are we justified in adopting this method of reckoning against such preponderating evidence to the contrary in this age of enlightened and logical therapy?

The commonly accepted symptoms of cinchonism may and do cause us to err in our efforts to cinchonize our patients by the stomach, for of tinnitus aurium, nausea, relaxed and moist skin, dilated pupils, generally accepted as indicating quinia in the blood, the two former are most depended on and least reliable. In fact, I do not believe them due to quinia in the blood at all, but to the application of quinia to the end organs of the nerves in the stomach mucous membrane. They are like the urticaria due to gastric irritation, a manifestation of the irritation of those nerves by quinia. Not infrequently violent complaint of those symptoms has

been made during the stomach administration of quinia and by changing to the hypodermic method, even with an increased dose, the complaint has ceased. You will pardon a brief report of a case in this connection which illustrates the fact. Mrs. C., a malarial cachectic of repeated recurrent attacks during the last 12 months, was suffering quotidian malarial paroxysm. Quinia sul. was prescribed morning and night in gram doses after Koch's method. The paroxysm occurred next day at the usual hour. Quinia was increased to three grams daily; the next morning the paroxysm was on time and hemoglobinuric. She was complaining vehemently of cinchonism and insisted that no more quinin be given, as it was doing no good, and making her sick. Now, one who in the least feared quinia as a cause of hemoglobinuria would have discontinued it when the disease assumed the hemoglobinuric form under 45 gr. doses, but the method of administration was changed to hypodermic and 20 grs. given every 4 hours, or 100 grs. per day, and the symptoms of cinchonism largely disappeared, the urine cleared, and the patient made rapid recovery.

In this case, as in several others equally as emphatic, the symptoms of cinchonism complained of disappeared under the increased dosage when the drug was removed from the mucous membrane of the stomach, and what some would have considered hemoglobinuria produced by quinia, disappeared under vastly increased doses when it was put directly into the blood. The only rational conclusions to be drawn from such cases are, first, the quinin applied to the gastric mucosa produced the symptoms called cinchonism and that its removal permitted the symptoms to subside; second, that the quinia had never been absorbed and carried into the blood, and therefore had not killed the parasite, whose ravages continued till quinia was so applied and terminated their career.

Summing up the evidence for and against quinia as a cause for this disease, we have:

Of the physicians reporting, 29.4 per cent. affirm it.	Of the physicians reporting, 70.59 per cent. deny it.
When quinia is suspended in treatment, 73.8 per cent. recovered.	When quinia is administered 83.1 per cent. recovered.
Distinct recurrences of attacks after the first appearance of black water in non-quininised patients, 9.8 per cent.	Distinct recurrences of attacks after the first appearance of black water in quininized patients, 4.4 per cent.
Per cent. of cases occurring from quinin alone, no per cent.	Per cent. of cases occurring from malaria without quinin, 15 per cent.
Per cent. of cases where quinin was thought to aggravate, 5.9 per cent.	Per cent. of cases where quinin was thought not to aggravate, 55.4 per cent.

With such preponderating evidence against quinia as a causative agent, that portion of the subject is left with you.

TREATMENT.

In taking up the treatment of this disease I shall be as brief as possible, giving the greater time, first, to those methods on which we differ; second, to those thought most important; third, those not in common use—dwelling but little on those measures of common acceptance and usage among us.

The inter-relation of cause and treatment, or the dependency of the one on the other, has caused in considering the first the foreshadowing of the most important feature of this portion of our subject. If the disease is due in part or whole to malaria, the treatment should be to remove the cause by administering quinia, the specific, in a manner sure to reach the parasite in the blood, and in sufficient dose to kill with certainty.

In dangerous diseases like this we should never reverse the order of the old Latin maxim, *"curare, cito et jucunde." "Curare"* should stand first; and in selecting methods of administration the one surest to carry the remedy to the parts desired should without exception be our choice, so long as imminent danger to life is not incurred—hence, intravenous or hypodermic administration should be resorted to in medicating a patient suffering from this trouble.

In dosage of quinia one should steer well on the side of the drug, remembering that the parasite is of that

type most resistant to quinia, and as a rule has become to some extent habituated to the drug. Bearing these facts in mind and taking for our base the dose that ordinarily proves efficient in other forms of malaria, an average of 30 grs. daily, we would take for an average dose in this condition, 40 grs. intravenously or hypodermically per day, until the next period for an exacerbation is safely passed, and continue it in half that dose until the second expected exacerbation is safely passed, when it should be discontinued for three or four days, and repeated in 20 to 30 grain doses every four to six days till five or six weeks are passed without a paroxysm. In the primary administration of the remedy one should remember that the older the parasite the greater its resistant power to quinia; therefore the sooner it is administered after the parasite has sporulated (the onset of the paroxysm) the easier it is killed and the surer is one to prevent the next paroxysm, which in pernicious malaria frequently means death to the patient.

Since Ivanoff,[1] by a careful study of the effects of quinia and methylene blue on the malarial parasite, has proven that quinia has its greatest affinity or toxic effect for the chromatin or nucleus of the plasmodium, which constitutes nearly the whole of it soon after hatching, and loses it as the achromatic zone accumulates around the nucleus as the parasite grows older and larger (a fact well proven clinically long since); and that methylene blue has greatest affinity for the achromatic zone (nicely portrayed by Romanovsky's method of staining), it behooves us to remember methylene blue in cases where we are called so late that quinia alone might fail to prevent the pending paroxysm, and to combine then the methylene blue in 3 to 6 grs. doses, two or three times daily. Another characteristic of methylene blue that recommends it in these cases is its sedative effect on the nervous system as portrayed by Hughes

1. Deutsche Med. Wochenschrift, May 2, 1901.

and Lovelace,[2] where of 22 cases of acute mania treated with it 16 received natural refreshing sleep. Of four cases of this disease reported treated by methylene blue there were three cases, or 75 per cent. recovered. Of the 19 cases reported treated with quinia hypodermically only one death is reported, and that occurred within 48 hours, showing that it was the result of the paroxysm had prior to any treatment, and not after quinia or any other remedy might have prevented it.

The tardiness of the effect of arsenic as an antiperiodic practically puts it out of consideration in conditions so pernicious, and its tendency to irritation of the renal epithelium also contraindicates it. There is no other drug whose mention in this connection is worthy of our time.

Considering other conditions for treatment, the inactivity of the liver stands first and a cholagogue is certainly indicated, for the presence of free hemoglobin in the plasma would alone, if undisturbed, soon prove fatal, to say nothing of the stagnant portal circulation obstructing nutrition and medication intended to alleviate other conditions. Calomel is certainly one of the best, if not the best, cholagogue and its inoffensiveness to the mucous membrane of the stomach commends it highly in this trouble where nausea is a predominating feature, hence calomel in small doses, often repeated (¼ gr. every half hour) is a favorite and justly so, and I would add 1-20 gr. of hyd. bichlorid hypodermically every five hours till the liver is well aroused, with massage of the liver or mustard plasters over it to the point of thorough redness of the skin. Of the 200 cases reported all save one were treated with calomel as a cholagogue and I note that the greater one's experience with this disease the more urgent is his advocacy of calomel.

Certainly to unload all effete material from the bowels and stimulate their eliminative function would add to

2. Phila. Med. Jour., March 22, 1902.

Daily dose of quinia; how administered	20-30 gr. Stomach				30-40 gr. Stomach				40 gr. and up. Stomach				20-30 gr. Hypod'rmically.				30-40 gr. Hypod'rmically.				40 gr. and up. Hypod'rmically.				15 gr. or less, or no quinin.				15-20 gr. Uncertain.				Methylene blue.			
Manner, cause and time of death	Uremia within 48 hours.	Toxemia within 48 hours.	Uremia after 48 hours.	Asthenia after 48 hours.	Uremia within 48 hours.	Toxemia within 48 hours.	Uremia after 48 hours.	Asthenia after 48 hours.	Uremia within 48 hours.	Toxemia within 48 hours.	Uremia after 48 hours.	Asthenia after 48 hours.	Uremia within 48 hours.	Toxemia within 48 hours.	Uremia after 48 hours.	Asthenia after 48 hours.	Uremia within 48 hours.	Toxemia within 48 hours.	Uremia after 48 hours.	Asthenia after 48 hours.	Uremia within 48 hours.	Toxemia within 48 hours.	Uremia after 48 hours.	Asthenia after 48 hours.	Uremia within 48 hours.	Toxemia within 48 hours.	Uremia after 48 hours.	Asthenia after 48 hours.	Uremia within 48 hours.	Toxemia within 48 hours.	Uremia after 48 hours.	Asthenia after 48 hours.	Uremia within 48 hours.	Toxemia within 48 hours.	Uremia after 48 hours.	Asthenia after 48 hours.
Deaths with hemorrhage from kidneys alone	.	1	7	2	.	.	1	1	.	.	.	2	.	.	.	.	.	.	.	.	.	.	.	.	3	1	1	2	.	1	1	3	.	.	1	.
Deaths with hemorrhage from other organs and kidneys	.	.	1	.	.	1	.	.	.	.	1	.	.	.	.	.	1	.	.	.	.	.	.	.	1	3	3	2	.	1	3	1	.	.	.	.
Recoveries with hemorrhage from kidneys only	37				4				30				8				4				3				36				9				3			
Recoveries with hemorrhage from other organs and kidneys	2				. . . . .				2				. . . . .				. . . . .				3				9				. . . . .				. . . . .			
Total number of cases treated	50				8				35				8				5				6				61				19				4			
Number and per cent. of recoveries	39 cases or 78%				4 cases or 50%				32 cases or 91.4%				8 cases or 100%				4 cases or 80%				6 cases or 100%				45 cases or 73.7%				9 cases or 47.4%				3 cases or 75%			
Number and per cent. of deaths	11 cases or 22%				4 cases or 50%				3 cases or 8.6%				. . . . .				1 case or 20%				. . . . .				16 cases or 26.3%				10 cases or 52.6%				1 case or 25%			
Per cent. of deaths in quinin treatment by stomach and hypodermically	18 cases or 19.3 per cent.												1 case or 5 26 per cent.																. . . . .				. . . . .			
Per cent. of deaths in each treatment	19 cases or 16.9 per cent.																								16 cases or 26.2%				. . . .				1 case or 25%			
Number and per cent. of cases with hemorrhage from other organs	9.8 per cent.																								29.5				. . . . .				. . .			
Average duration of illness	Treatment with quinin—5 days.																Treat't without quinin—9.66 days.																			
Opinions of reporting physicians	70 6 per cent believe quinin does not produce hemoglobinuria.																29.4 per cent. believe quinin does produce hemoglobinuria.																			

the chances of our patient, and enemas of hot solutions of sulphate of magnesium accomplish this quickest and surely, together with stimulating diaphoresis, a valuable means of eliminating much undesirable material from the blood and relieving the irritated kidneys of some tax. To allay the nausea and vomiting, iced champagne has proven most efficient in my hands, with ice cold beer a close second, the former adding stimulation and the latter both stimulation and diuretic effect to its credit. While large drafts of very hot water have often proven very efficacious as a stomach sedative, as well as a diaphoretic and stimulant, one of my advisers strongly urges this as the most important treatment of all. So soon as the stomach is in a condition to appropriate it, nutritious fluid food should be given at regular and proper intervals. For the kidneys, hot fomentations over the loins or perhaps counter-irritants with mild diuretics should be given, every effort being made to save the kidneys any irritation and labor. The intravenous injection of large quantities of normal salt solution serves well to stimulate over the depression, and assists in eliminating poisons from the blood, and in one case reported, where entire suppression of urine had occurred, it started up the renal secretions and the patient recovered when apparently he was doomed. Stimulants and tonics hypodermically in liberal doses should be used to maintain the vital functions, and here strychnia stands first. So soon as the expected paroxysms are safely passed, ferruginous and bitter tonics per os are to be given, steam baths and sweat baths are recommended and have to commend them the greatest relief of the kidneys by their diaphoretic action.

The time and space required to enumerate writers and practitioners who have assisted in these efforts are too great to allow their recital, hence the omission of a bibliography.

DISCUSSION.

Dr. William Krauss, Memphis, Tenn.—One can not study diseases by any rules of logic, but we can discuss that way. The reader of the paper states that, among about 170 cases reported, the blood examination was made in 12, and 7 of these 12 did not show the malarial parasites; if that were so, I should like to know why he cinchonized these 7 patients? About 12 years ago I attempted, by proxy, to study this disease and found in a very short time that it was exceedingly unsatisfactory. Then I attempted to place myself in touch with gentlemen in the vicinity of Memphis, requesting them to wire me when they had such cases and I would take the first train to them. This also proved to be unsatisfactory. I then decided to abide my time until I could get a visiting service in a local hospital where I could study the disease properly from beginning to end. I think it is illogical to attempt to classify the mortality according to the treatment, irrespective of the pathologic conditions; whatever remedy we use should be given to overcome the pathologic conditions or to remove the etiologic factors which may be present in a given case. Judging from the character of the cases reported he evidently saw cases of the intermittent variety, accompanied by chills and fever, and such cases are exceedingly rare during the season of the estivo-autumnal parasites. They are found in the spring and early summer and are very transient. In his cases he found the parasites persisting through the attack. It is proper to cinchonize such cases.

What is the pathologic condition to be found in such cases? It is a steatosis of the kidneys and liver, a rapid breaking down, and, if present, a disappearance of the malarial parasites. The treatment must be directed to that end. What is the pathology of the blood? There is a hemolysis. If you have studied the subject of hemolysis only very superficially you know that it requires a specific immune body and a complement to produce it. The normal mechanism of this in malarial infection is probably for the destruction of infected red blood cells. There is a later storage of a surplus of this specific body; the complement may be produced either by quinin or a new superadded infection—either, then, may be the occasion of an attack. This is where the question of administration or non-administration of quinin comes in. In the majority of the cases the hemolysis effects the destruction of the parasites. The subsequent temperature may be a guide; as a rule, the present malarial infection may be ignored. The doctor has stated that only small doses of quinin produce the hemoglobinuric fever, and in one case he produced it with 90 grains; if that is a small dose I should like to know what he considers a large dose.

Last summer, during my illness, there were admitted to St.

Joseph's Hospital 21 cases of malarial hemoglobinuria, and all were treated with hypodermic injections of quinin; two recovered. On the other hand, I have now an unbroken series of 21 cases without a death. The main point is, avoid cinchonizing these patients. Take such measures as you can to prevent fatty changes taking place.

Dr. Frank A. Jones, Memphis, Tenn.—A few days ago I heard Dr. Osler say: "Tuberculosis is the captain of the men of death." I should like to paraphrase that expression, and say: "Tuberculosis is the captain of death and malaria is his first lieutenant."

We have in our midst to-day a quartet of men who have been working in malaria. One of these is Dr. Krauss, and there is no man in this country who understands malaria better than he. Then we have among us another gentleman whom we all know and greatly respect, and who has written a book on malaria; I refer to our chairmain, Dr. W. S. Thayer. Again, we have another gentleman of brawn who has been brought up in a country district, at a little flag station, but yet who has the best equipped laboratory in Mississippi; I refer to our secretary, Dr. James B. McElroy. One is a wheelhorse; the other an Ajax. Then, too, I ask that Dr. Sutherland of Mississippi, another man who has done much on this subject and a man whom all greatly respect, be asked to address this Section.

Dr. H. L. Sutherland, Rosedale, Miss.—This subject has been frequently discussed and the principal feature of discussion always is as to the advisability of the use of quinin. I think there is one proposition on which we all agree, namely, that in the presence of an active estivo-autumnal infection we should use quinin. It seems to be the general opinion that "the existence of the hemoglobinuria itself, or the presence of some other toxic substance in the blood" (Thayer) destroys the parasites and renders the administration of quinin unnecessary and therefore harmful; but, in my observation, this is not always true. The use of the microscope should then govern, in these cases, the use of quinin. But, unfortunately, only a very small percentage of physicians practicing in sections where hemoglobinuria occurs have a practical knowledge of the use of the microscope, and therefore must find some other method of determining the treatment.

After the appearance of hemoglobinuria, my first attention is directed to elimination, and I do not give quinin in the first twenty-four hours; if the fever continues high, I give 15 or 20 grains of quinin or two ounces of Warburg's tincture. If the patient has taken quinin preceding the attack, I am not in a hurry to give more, but rather await the result of what he has taken. I can better illustrate by citing two cases in point: In August, 1901, I saw a case with a temperature

of 101, and next day 102, and the third day 104.8, when I gave two ounces of Warburg's tincture, and his temperature dropped to 101. I repeated the dose and his temperature became normal, and on the fifth day of the disease there were a few estivo-autumnal organisms present.

Oct. 25, 1902, my son had a chill and in the next forty-eight hours took 48 grains of quinin. Several hours after the last dose he had a chill with fever, which soon subsided and I gave no more quinin; in thirty hours after the last dose of quinin he had another chill with hemoglobinuria, but his temperature did not go over 102, and in another twenty-four hours his urine had cleared and temperature was normal. I supposed he had had sufficient quinin prior to the hemoglobinuric attack to kill the malarial organisms. A blood examination made by Dr. Krauss of Memphis, October 31, showed "very active estivo-autumnal infection," and I began the administration of quinin again. These two cases prove, to my mind, that in some cases at least quinin should be given, while, perhaps, in a majority of cases, they get along better without it; and I have adopted the rule that where the fever following hemoglobinuria continues high after tewnty-four hours, or from a temperature as low as 101, its tendency is to go higher each succeeding day, I give quinin, and following this rule I find I give it in about half of my cases.

Dr. James B. McElroy, Stovall, Miss.—I agree with the reader of the paper in the statement that hemoglobinuric fever is, in the majority of cases, due to malarial infection. That parasites are not always found in the peripheral circulation is due to several circumstances. Stephens and Christopher think it is due mainly to the fact that quinin has usually been administered previous to the occurrence of black water. This is undoubtedly true, but I have seen at least one case in which parasites were present previous to the attack; no quinin was administered previous to nor during the course of the disease, and the parasites disappeared a few hours after the onset. I have surmised that this is due to the fact that in the extreme hemolysis which takes place the cells of low resistance will naturally be the first to be destroyed, consequently the cells containing parasites are usually first destroyed. The parasites, being set free in the plasma, soon die.

I have observed more than fifty cases of this disease and in 19 cases have made repeated blood examinations. In only 5 of these have parasites been present, estivo-autumnal in 4 and double tertian in 1. The malarial nature of the remaining 14 was demonstrated by the leucocytic variation and pigmentation characteristic of malaria, and by the character of the febrile attack previous to the development of black water.

As to the relation of quinin to hemoglobinuric fever, in a few instances black-water fever develops in the course of a

malarial infection in which no quinin has been administered, as in the case just referred to. But in the majority of cases it occurs in the course of an old malarial infection in which quinin has been administered. And my experience is in accord with the findings of the malaria commission to the royal society, which concludes that black-water fever is of malarial origin, but that it is not a malarial attack *per se;* that in the majority of cases quinin is the immediate causative agency. I have never seen a pure quinin hemoglobinuria, though it undoubtedly has occurred.

The few experiments which have been made *in vitro* to determine the hemolytic effect of quinin in these cases have, so far as I know, been negative. Recently I have been making some experiments along this line which, though limited, indicate that quinin has a hemolytic action on the blood of these cases.

As to treatment, occasionally in cases of intermittent hemoglobinuria—and I may say that such cases are comparatively rare in my experience—quinin used cautiously may be of benefit. But as a rule in the cases of continuous hemoglobinuria I do not think that quinin should be administered. In the first place, it is not indicated, as the parasites are not usually present, and when present its administration has aggravated the condition in my experience, as the two following cases will show: In the first case of the series of the 19 cases referred to above, parasites were observed during the course of the disease, on the third day when the urine had begun to clear. Following Bastianelli's rule, I determined to administer quinin. Seven and a half grains were given hypodermatically and four grains ordered per os every four hours. Two hours after the first dose the urine became black, suppression and death following during the night. Whether this was due to the sporulation of a new group of parasites or to the quinin administered, I am unable to say.

In case number ten of the series, parasites were observed during the course of the first day of black water when the urine was clearing. Quinin was administered and a few hours afterward black water returned with an aggravation of all the symptoms. Quinin was immediately discontinued, and the patient recovered.

Dr. W. S. Thayer, Baltimore—There is little of value that I can say which has not already been said by those who have had much more experience with this condition than I. Hemoglobinuric fever does not occur in Baltimore. Those cases that I have seen I have visited at distant points—in Virginia, Tennessee and North Carolina. My knowledge on the subject has been obtained mainly from literature. One should remember that hemoglobinuria is not a symptom proper of malaria; it is a condition for which malaria opens the way; its remarkable limitation to special malarial districts is also well known.

But one should further remember that hemoglobinuria occurs as well in a variety of other infectious diseases. It may complicate typhoid fever, yellow fever or smallpox. In malaria it may occur at the height of an acute paroxysm when parasites are present in the circulation. In many cases, however, the malarial attacks disappear on the first outbreak of hemoglobinuria, and parasites can no longer be found in the blood. Again, the condition occurs in patients who have recovered for a greater or less length of time from their acute malaria and are in a condition of postmalarial cachexia. This is often the case. In certain parts of this country the disease is met with more frequently in the late fall, at about the time of the beginning of frosts. I believe that it is fair to state that, in the majority of cases, within twenty-four hours of the onset of the attack of hemoglobinuria, the parasites of malaria are not to be found in the blood. If the case, however, be left to itself relapses of the malaria will follow sooner or later in many instances.

Quinin is of no more value in the treatment of the hemoglobinuria as such which occurs in connection with malaria than it would be in hemoglobinuria connected with such diseases as yellow fever or typhoid fever or smallpox. Quinin is of value in removing the ultimate cause—the malarial parasite; it is of no value in the treatment of the immediate conditions on which the hemoglobinuria depends. If the parasites do not disappear from the blood it seems to me that we are bound to administer quinin. If, in a given case, one suspects that the patient has pernicious malaria and microscopic examination of the blood is impossible, quinin should certainly be given at the onset of the trouble. If, however, after twenty-four hours no parasites are present in the blood it may be better to postpone quinin treatment until later.

With regard to the deleterious effects of quinin: True quinin hemoglobinuria must be of the greatest rarity in this country. Cases have been reported, especially in Sicily, in which the most minute doses of quinin have produced violent attacks of hemoglobinuria, cases in which there can be no doubt as to the nature of the condition. These instances depend probably on individual susceptibility.

With regard to the method of administration of the drug it is hardly worth while to speak. Quinin acts most promptly when given intravenously, and the dihydrochlorate gives excellent results when thus administered.

Dr. Walter Shropshire, Yoakum, Texas—With regard to the assertion by Dr. Krauss that I studied hemoglobinuria by proxy, I want to say that no one examined these cases but myself. I reported 29 cases in the series, and I had some I did not include because many of them were rather indefinite—I could not give in them a fair and honest consideration of the subject.

Among the 29 cases there were but 2 deaths. In one of these a physician asked me to attend a patient for him, but refused to allow me to treat the case in my own way. This patient had a mild paroxysm every morning for three mornings and then hemoglobinuria. I had promised this physician to give the patient calomel and withhold quinin until it acted. I saw this patient at 4 p. m. one day and I realized the mistake in giving him calomel and not quinin till after it acted, for scarce time for the calomel to act remained before the next paroxysm was due, but I had promised to treat the case according to the physician's expressed wishes, and I gave him calomel. Of course it did not prevent the next paroxysm. When I returned home I asked that physician never to send me to another of his cases instructed. The next morning this patient had another paroxysm, entire suppression of urine and a fatal end followed.

The other fatal case was one in which there was a distinct nephritis present. He died on the sixth or eighth day in spite of the use of quinin. Thus out of 29 cases only 2 deaths were recorded, and one due to the non-use of quinia.

The pathology of the disease has been overlooked. Ewing reports examinations of kidneys after death in one case, and he said he was unable to find or demonstrate the parasite in the peripheral circulation, but at autopsy the kidney capillaries were found to be filled with them. In every case at autopsy in the pernicious form the parasites are found in the spleen and the brain in comatose forms, and stomach and intestines in the choleric or enteric forms.

If the parasites are to be found congregated in such situations in forms when symptoms are referable to it, why not in the kidneys in this form? The fact is they have been where autopsy was had and adequate examination made in reference to the non-appearance of the parasites in the peripheral blood. I should like to refer the secretary to one of his own cases, where it was found on the third day that the peripheral blood contained them after repeated negative examination. It has been noted that in the pernicious form of the fever the parasite seems to leave the peripheral circulation and invades the central circulation and organs, hence we can not depend on examination of the blood from the peripheral circulation as a guide to our therapy in this form of disease.

SOME CLINICAL ASPECTS OF TUBERCULAR PERITONITIS.

JOSEPH EICHBERG, M.D.
CINCINNATI.

Among the many startling results that have followed the introduction of laparotomy, exploratory or curative, none shows a more decided departure from beliefs of a former generation than the present attitude of the profession with regard to tubercular peritonitis. The surprising clinical fact, recorded by Sir Spencer Wells, that a case of tubercular peritonitis, accidentally opened under a mistaken diagnosis of ovarian tumor, should not only recover from the immediate effect of the operation, but promptly go on to apparent recovery, and end in perfect restoration to health was so anomalous a history that it savored of the miraculous. This was before the days of bacteriology, and while the battle as to the single or dual nature of tuberculosis was a great pathologic issue. With the knowledge of the bacillus as the one essential feature in causation, and with the great advance in surgical technic, the pathologic relations, as well as the clinical features, have received a more critical study, and much light has been gained.

Brunn[1] gives a most comprehensive review of the literature of this subject from 1885-1900. Our knowledge may be said to have reached its greatest development in this period. Many of the tables and references are taken from an analysis of this review. Of some 28,000 cases coming to autopsy, many of these were healed,

1. Cent. f. allg. Path. u. Path. Anat., 1902.

many showed co-existing diseases of other organs. Tubercular peritonitis was found in 2¾ per cent. As early as Morgagni we find descriptions of tubercular peritonitis, with the significant statement that it frequently occurred with pulmonary tuberculosis, and a suggestion of the similarity of adhesions here formed with those observed in the pleuræ and pericardium. Almost a century of study has thus been given to this particular form of tubercular disease.

The peritoneum may be one of the sites of local manifestation of a generalized miliary tuberculosis, but the term tubercular peritonitis is generally understood to apply to the chronic form of the disease, which in a measure constitutes a distinct affection. Even here the process is usually secondary to disease, appearing primarily in some distant part, most commonly the lungs; or the peritoneal involvement may follow by direct extension or by dissemination through the lymph paths, or by a simple process of grafting from a previously ruptured tubercular focus having access to the peritoneal sac, and sowing, as it were, the elements of the disease over the entire membrane.

As primary foci near the abdominal cavity may be mentioned psoas abscess, caries of the vertebræ, tuberculosis of the genitourinary tract in both male and female, including the suprarenal gland, tubercular ulceration of the intestine, solitary tubercle of the spleen, and tubercular disease of the retroperitoneal glands. Some cases of tubercular peritonitis appear to be primary, as shown partly by the manifestly greater age of the diseased parts of the peritoneum when contrasted with the evidence of tuberculosis in the other organs, in part by the absence of any other discoverable tubercular deposit. In children the appearance of primary tubercular peritonitis is more frequent than in adults; of 22 cases reported by Rilliet and Barthez, 12 were classified as primary.

Tubercles may be distributed uniformly, more or less

thickly set, over the entire peritoneum, or they may form nodular masses of varying sizes which undergo caseous or ulcerative changes, and thereby furnish the nucleus of secondary daughter tubercles in the vicinity.

With such multiplicity of lesions, and with the symptoms of the primary pathologic process complicating most irregularly the clinical picture of the peritoneal affection, it is not surprising that there should be a wide range in the symptomatology. On the basis of the postmortem findings various classifications have been made. The simplest recognizes two forms, the dry and the exudative, including under the latter head the caseous and the purulent varieties.

From a clinical standpoint Hawkins divides all cases into four varieties:

1. Latent.
2. Severe, with ascites and a spontaneous tendency to remissions or encapsulations.
3. Cheesy purulent form, with frequent formations of large caseous masses and adhesions.
4. Fibrous-adhesive form.

The French and English authorities generally describe three varieties:

1. Ascitic.
2. Fibro-adherent.
3. Ulcerative form with or without suppuration; while Galvani gives five, and Aldibert has some eleven subdivisions.

Judging by inoculation experiments, differences in the pathologic product of the bacilli depend in some degree on the source of infection. Injection into the peritoneum made with fluid obtained after weakening of the bacilli in sputum by exposure to a temperature of 60 degrees, produces rather the fibroid; the use of a fluid rich in bacilli or of unemulsified sputum, the cheesy, purulent variety.

Injection of the first tuberculin of Koch produced no

reaction, whereas the tuberculin R caused proliferation of the intestinal serosa.

The general symptoms of tubercular peritonitis are pain, fever, emaciation, anemia, anorexia, to which are to be added as more purely local manifestations, change in the contour of the abdomen, ascites, diarrhea, or constipation; and, occasionally, redness or inflammation about the umbilicus, with possible perforation and formation of a suppurating sinus or of a fecal fistula; profuse and very exhaustive sweats occur in the caseous and ulcerative varieties. Of physical signs, the most important may be properly said to be the alteration in the impressions conveyed by palpation, whether in the dry or exudative cases. In children, particularly, the examiner is apt to be impressed by the manifest malnutrition, so that there is a striking contrast between the distended abdomen and the general emaciation. With the patient on the back and the abdomen relaxed to the utmost possible extent, there will usually be noted an oval or round distension of the cavity, very different from that due to ascites, arising from other causes. There is not the tendency to bagging in the groin, or above the iliac fossa. If liquid be present, it is rarely perfectly free in the abdominal cavity, being bounded in one or other direction by firm adhesions. After absorption of this fluid the exploring hand experiences a sense of tension or hardness in the abdominal wall, a peculiar elastic resistance, which, according to Grisolle, is found in no other malady, and which has been described by DeMussy as an incomplete elasticity with resistance, as though the hand were plunged into firm dough.

In addition to this the abdominal wall may, over certain areas, give an impression of plaque-like thickening, there being in these places increased induration and resistance. Palpation should always be made by laying the whole hand gently yet evenly on the abdominal wall, and then carefully indenting the fingers, preferably

moving all the fingers together, so that no indentation of the walls occurs at any one point. In this maneuver it will occasionally be found that adhesions and loops of intestine, particularly when distended, may simulate tumors, especially in the region of the cecum. Occasionally loops of intestinal coils may be seen and felt through the thinned muscular wall; this is exceptional. Palpation will also show the presence of fluids, which may or may not obey the law of gravity with the patient's change of posture.

Attention has already been directed to the cases with exudation as constituting a special class; yet the value of such classification may well be questioned, when it is remembered that in very many cases the presence of abundant fluid constitutes rather a phase than a form of the malady. In children, for the most part, and in many adults, the so-called ascitic form is only transitory, and the fluid is later resorbed; so that the value of effusion as a symptom may depend on the period at which the case is seen. It may even happen that as the effusion disappears, the contour of the abdomen changes, though a permanent meteorism usually occupies the space occupied by the fluid. Thomayer has called attention to a physical sign of some value. In the shortening of the mesentery resulting from tubercular infiltration, retraction takes place toward the base of attachment, so that the entire mass of intestinal coils, when not previously adherent, is drawn upwards, backwards, and to the right, while the fluid exudation accumulates below, and to the left.

By this change the symmetry of the abdomen is often disturbed, as may be verified by inspection, palpation and percussion. Palpation will also disclose the presence of the transverse induration about or just below the level of the umbilicus, due to the infiltrated and curled-up omentum. This sign is wanting where, as often happens, previous adhesions have fixed the omentum to some of the pelvic viscera or to its brim. Where tu-

bercular ulceration exists, particularly if multiple, movable cylindrical masses, in reality the hose-like, greatly infiltrated intestine can also be outlined. In some instances a distinct friction rub, either localized or more general, will be noted; it may be a very mild friction, what the French writers term starch-like, *amidonienne,* or an unmistakable harsh, rasping rub. Auscultation may be employed to control or check the results of palpation. During palpation one observer describes the condition as suggesting that the abdominal wall and subjacent viscera, firmly united by adhesion, could not glide over each other. The friction rub is felt more frequently in the upper quadrants. Percussion corroborates the presence of the fluid, and its data should be carefully noted. It will inform us as to whether the fluid is movable or sacculated in small pockets, as often happens; according to Grisolle, in one-tenth of the cases.

A careful record of the areas of dullness should be kept from day to day, for, as Christie has remarked, a striking feature consists in the atypical character of the percussion data, changing slowly and imperfectly with change of posture, but often varying spontaneously. It can usually be determined that the quantity of the fluid is inconsiderable; it may amount to 2 or 3 liters, but never equals the enormous amounts encountered in cases of obstructive lesions of the portal vessels.

Of the general symptoms fever is perhaps the most inconstant. It may be encountered at one stage of the disease (usually in the earlier periods) to disappear for many weeks or months and return when ulceration takes place in some of the caseous masses. The number of observations on the temperature is not very extensive, yet enough has been collected to show the frequent absence of fever in cases where the diagnosis has been confirmed by autopsy or by laparotomy. It has been absent in the majority of my own cases. In 16 cases observed by Hinterberger there was fever following the operation in 14; five times it had existed before, 9 times

it developed for the first time after operation, and two cases remained afebrile throughout. In several cases occurring in my own practice, other physicians, while inclined to the diagnosis of tubercular peritonitis, had found the absence of fever an insuperable barrier to such a conclusion. High fever usually accompanies ulcerative tissue destruction. Pain may be entirely absent. It occurs in the majority of all cases; it is oftentimes constant and fixed, though it may change both in intensity and location. Its development is often very gradual, consisting at first in a sense of uneasiness, which becomes accentuated where any weight rests on the abdomen, and is certainly felt on pressure. Despite the fixity of the lesion, the pain may be erratic, and its maximum intensity may not correspond with the location of the trouble. When not spontaneously present it may often be developed by suddenly releasing the abdominal wall, previously depressed by firm and gradual application of the flat of the hand, as suggested by Henry. In adults persistent abdominal pain, with no other symptom than progressive emaciation, would suggest a possible tubercular peritonitis. The tenderness over infected viscera may become extreme.

Diarrhea is the regular accompaniment of tubercular ulceration of the intestine, but in other forms constipation is the rule. There may be but one movement daily, but the dejections are liquid, and this fact may be utilized in diagnosis. In children the stools are often acholic (Berggrün and Katz), though there is no jaundice or bile-coloring matter in the urine, the clay color of the stools being due to excess of fats. On the other hand, the constipation may be most aggravated, and may go on to complete intestinal obstruction, due to the pressure of strands of adhesion, twisting or paralysis of the bowels, or the formation of large tumor masses in which intestinal coils become adherent. The obstruction usually ends by yielding either spontaneously, or with

medication, after some days. Death has resulted, the end coming slowly after twenty to thirty days. Anorexia is frequent; vomiting, especially in children, is very rare.

Periumbilical inflammation may occur in other forms of suppurative peritonitis, but is most frequently due to tuberculosis. From this hasty summary, it will appear that there is no absolute sign that clears the diagnosis, which is often to be made only by exclusion. Age has a most important bearing. While exceedingly rare in children under one year of age, one case only in 300 collected by Carr, the early periods of life contribute the largest contingent, a fact frequently overlooked because of the tendency to spontaneous cure; it is infinitely the most common form of peritonitis between the ages of six and ten, and, though beginning acutely, may soon become subacute and thus continue throughout.[2] Spontaneous cure has baffled many students; yet no one would to-day repeat such a criticism as this of Siredey: "We believe that Hilton Fagge suffers from a strange illusion when he says that the majority of children suffering from tubercular peritonitis may be cured by mercurial liniments, and we hasten to add that such observations as he has cited in support of his views are absolutely valueless." It is difficult to divorce from the professional mind the idea of necessary fatality in connection with tuberculosis of any internal parts.

In doubtful cases with ascites, the withdrawal of fluid has been recommended for agglutination and inoculation tests. Benedix claims to have found a positive agglutination test in a dilution of one to ten, while a non-tubercular ascites failed; both conclusions were corroborated by autopsy, the second case showing cirrhosis. The inoculation test in lower animals would, however, appear to be the main reliance. Bacteriologic examination of the fluid withdrawn may show nothing, yet the same fluid will promptly give rise to an inoculation

2. The proportion of the sexes is nearly equal before ten years, but the female sex furnishes a much larger contingent after this.

tuberculosis in guinea-pigs. A most important factor in diagnosis is the coexistence of tuberculosis in other localities, particularly tuberculosis of other serous membranes. Aran has formulated his view that tuberculosis of the peritoneum often is a part of a more generalized process affecting serous membranes. Goddier says: "Where there is tuberculosis of the peritoneum, there is always tuberculosis of one or both pleuræ as well," and other observers have directed attention to what is termed an isolated tuberculosis of serous membranes. As the starting point for this form of tuberculosis is often found in the genital tract, a very careful examination of these organs is essential. In women nodular masses of varying sizes will frequently be found in and about the pelvis.

Laparotomy does not always establish the diagnosis. Even with the entrails exposed, error is possible. Echinococcus vesicles, the eggs of the tenia, and simple cell inclusions have all given rise to tubercular formations, whose macroscopic diagnosis was impossible. The nature of the fluid is often suggestive, the sacculation of the exudate, the whitish opacity or bloody tinge of the liquid with its viscid, jelly-like consistence, all argue for tuberculosis; absolute certainty is, however, only possible with the microscope or inoculation test; but tuberculosis may be generally assumed as the cause of nearly all nodular tumors, merely on the doctrine of averages.

We now come to the question of laparotomy. The example of Spencer Wells, of performing an operation under a mistaken diagnosis, for a condition which then promptly improved, has been unintentionally repeated several times, and probably will continue to be repeated. Too much credit for the recovery can not fairly be assigned either to the operation or the operator. König undoubtedly is responsible for the popularization of laparotomy as an element in the treatment; and since his day there have been many undoubted cases, as shown by autopsy or by survival for many years of healed tuberculosis following abdominal section. Indeed, some cases

are on record of coexisting tuberculosis of other organs, in which, following operation, these additional lesions presented notable improvement. Has this not been another instance of the *post hoc*? The operation is constantly urged by the surgeon, and Pribram goes so far as to advise its performance in every case, which shows no improvement after a few weeks of medical treatment, even where there is a pulmonary complication.

Good results have followed simple incision, irrigation of the cavity, drying of the peritoneum, resection of diseased parts, and other modifications. The essential feature seems to be an incision, even when not carried into the cavity; for there are many cases in which the cavity no longer exists. Some regard as essential the mere access of air, and Mosetig, to be on the safe side, merely punctured the abdomen, and then inflated it with sterilized air, thinking thereby to impart some altered quality to the bacilli. Then it has been held with unconscious humor that the opening of the abdomen allows of such rapid drying that the bacillus dies. Others regard the effect as due to more pronounced reaction of the serous membrane after operation. In cases with ascites the mere mechanical relief following withdrawal of the fluid can be easily understood; the diminution of pressure may also allow better conditions for resorption and improve the circulation. It is a widely entertained opinion that operation assists the peritoneum to more effective isolation of tubercular foci by stimulating the growth of new formed connective tissues. Another more modern theory would explain the action by the agency of antitoxins, whose formation is started by the operation, or of alexins generated in the blood; while another view attributes the improvement to the actual escape of the bacilli and their spores through the incision the moment they have a chance to get out!

After all this elaborate effort at explanation, the true answer to the question as to why laparotomy cures tuber-

cular peritonitis, is: "Because it does not." The percentage of recoveries without operation, for all ages, and with or without complications, is greater than with operation. This is admittedly true of all forms except that with effusion, and here the results of medical treatment have been most satisfactory; for these are the cases which tend most commonly to spontaneous cure. In the course of five and a half years Herringham observed twenty-five cases diagnosed as tubercular peritonitis, of which nineteen recovered without operation, and three were unimproved. He finds the only justification for operation is a return and increase of the ascites, after puncture. Hawkins reported fifty-nine recoveries, all but three of whom were treated by internal medication. From a collective report of 112 cases treated by operation, Hawkins concludes that the percentage of recoveries is as great without as with operation, the indication for which is furnished by the presence of marked ascites. The distinguished surgeon, Czerny, cautions against expecting too much from the operation, whose mortality in his hands has been very high, for out of ten cases operated on, but two survived after three months. Leyden asserts that operation furnishes no better results than can be secured without it. He attaches little importance to experimental inoculation or the detection of the bacillus, and thinks general symptoms sufficient for the diagnosis. Where abdominal tension from fluid is great, he recommends occasional punctures. According to Pic, the ulcerative form may heal in children, never in adults; the fibroid form heals in both, and the form best adapted for operation is tuberculosis with ascites. Wheeler declares that the operation is absolutely contraindicated where there is no exudation in the abdomen, or where there is abundant development of nodules and bands, or where there is marked tuberculosis of other organs, especially intestinal ulceration. With this view I am heartily in accord. Wheeler also advises against

drainage, owing to the likelihood of a fistula, fecal or tubercular.

The operation has been particularly recommended for exudative cases in children, but in the last number of the *Archives of Pediatrics*, Guthrie gives a summary of some personal experience showing recoveries with and without operation in the ratio of 16 per cent. to 50 per cent. His language in condemning the operation is not without a tinge of bitterness.

Jaffé has shown that many cases reported as cured still contained at the time when they subsequently came to autopsy, smoldering foci of tubercular disease, which required apparently but the merest accident for general dissemination, or for reappearance of the local process in active form.

The opinion that a diagnosis of tubercular peritonitis calls for immediate laparotomy is certainly erroneous, not to say vicious. It would be responsible for many deaths. In the hands of the most skilled surgeon the mortality incidental to the operation *per se* is not less than 10 per cent. Cases requiring relief by operation can be temporarily improved by simple puncture and withdrawal of the fluid. The majority of such cases under the tonic regimen will end in recovery without operation. It will be found that some cases will disappoint every expectation of improvement, under any plan of treatment, and again complete recovery will follow under conditions apparently most unpromising. What the particular factor is that determines the issue must still be doubtful, but increasing experience and most careful analysis of figures would lead us to place it in that unknown chemical and physiologic property, the native resistance of tissues. We may assume a variability in the toxic properties of bacilli, even as we see variations in structural details; and this variability plus the equally variable inherent constitutional resistance gives us our equation, whose resultant is naturally most inconstant. It is certain that the peri-

toneum, perhaps even in greater degree than most other serous membranes, possesses the property of finally disposing of the tubercle bacillus, by a process of fibroid inclusions, so effective that it not only results in a limitation of the infected area, but often in the actual destruction and disappearance of the pathogenic germ. We find ourselves coming back to the standpoint, which Virchow investigated in his studies of the "combat between the cells and the bacilli," and, while sparing no endeavor to limit the possibilities of entry of the bacillus, and to render innocuous those sources of infection easily controlled, it will be found that our best weapon lies in so fortifying the cells which, in the final test, must bear the brunt of the fight, that the natural processes of reactive inflammation may lead to the development of organized fibroid tissue. As in the fight against tuberculosis in the larger arena, so here the solution will be found in giving to the patient, in greatest abundance, good food, pure air, unbroken rest, and ease of mind. It is not that we would slight the surgical management of such cases; but surely that science is best which by early recognition of diseased conditions and prompt institution of appropriate remedies protects the patient from the necessity of submitting to the hazard of operation, with the possibility of no permanent relief. Immediate recovery from the effect of the operation can not be counted an advantage. It remains for collective investigation to show the percentage of patients in good health six months after the operation, before determining the position of laparatomy.

As illustrating somewhat different types, I append a brief history of the following cases:

CASE 1.—Discrete tubercles of peritoneum, probably secondary to appendicitis, with ulceration discovered at operation for appendicitis. Recovery.

S. R., aged 12. Has had repeated attacks of appendicitis, some very severe, others of less duration and intensity, coming on as often as every two months in the year preceding operation. Boy always pale, thin and feeble. Bowels habitually

constipated; no appetite. In intervals between attacks no tenderness of abdomen, no induration or sensitiveness anywhere about abdomen. Operation for removal of appendix in February, 1892. Appendix thickened, surrounded by adherent omentum and intestinal coils, small perforation with sacculated cavity containing a few drops of pus. Neighboring loops of small intestine, and parietal peritoneum showed limited number of gray nodules, firm, translucent, surrounded by a pigmented crescent, but apparently giving rise to little, if any, local reaction. Recovery after operation prompt. No subsequent symptoms except slight hernia in line of incision relieved by second operation. Young man to-day enjoys excellent health. There was no family history of tubercular disease; no signs of pulmonary or other involvement preceding operation.

CASE 2.—Generalized chronic tubercular peritonitis, with special localization about liver, producing chronic perihepatitis. Ascites. Repeated puncture. Death.

J. H. patient male, aged 54, single; generous liver. Family history good. History of persistent alcoholism, never carried to excess. Taken sick with sudden pain in abdomen, most marked in right hypochondrium; chills, fever of intermittent type; anorexia, constipation, gradually increasing distension of abdomen, dyspnea, emaciation, anorexia, subsidence of fever, but increase of abdominal swelling. No cardiac or renal disease; no jaundice. Physical examination shows abdomen distended with ascitic fluid, freely movable. Liver dulness not easily made out. Some tenderness or palpation over abdomen. After withdrawal of 2½ gallons of straw-colored fluid liver dulness found to be slightly increased. Abdominal walls apparently thickened and unusually resisting. No fever. Reaccumulation of fluid in very brief period. Death six months after onset of symptoms from exhaustion. Autopsy confined to abdominal cavity. Chronic perihepatitis (capsule thickened so as to measure half an inch), generalized tuberculosis, parietal peritoneum about one-quarter inch in thickness, intestinal coils showing few adhesions. Retroperitoneal glands enlarged, reddened, no tubercular infiltration. Diagnosis corroborated by microscopic sections.

CASE 3.—Dry adhesive peritonitis with caseous salpingitis. Operation. Final recovery after prolonged suppuration from tubercular sinus.

L. B., aged 21, dressmaker. Fairly well nourished, single. Family history, several deaths from tuberculosis in direct ancestry. Never been very robust, menstruation at 15, fairly regular, painful. Appetite poor, chronic constipation. No fever. Some pain in back and tenderness over abdomen. Physical examination, no pulmonary involvement. Abdomen oval, distended, rather resistant, no mass or induration. No

fluid. Diagnosis, tubercular peritonitis. Under general measures and attention to bowels some improvement. Four months later patient passed through attack of typhoid fever of moderate severity, during which persistent abdominal distension was most distressing symptom. Complete recovery from typhoid. Pain in abdomen dull, heavy and variable, continued. Operation. General matting together of all viscera; omentum stretched tightly in front had to be detached from borders of pelvic cavity, showing caseous tuberculous infiltration and softening of both tubes, several tubercular abscesses in pelvic cavity. Following operation patient has gained some in weight, the temperature slightly lower in range, but still persistent. Exudate still being poured out, however, showing the process still active.

Removal of adnexa, cleaning out of tubercular debris, irrigation and drainage. Tedious recovery and persistence of discharge from sinus left after removal of drainage tube. Irrigation of fistulous tract with zinc chlorid solution; no effect. Following use of small quantity of peroxid of hydrogen, marked and sudden increase of pain in abdomen with collapse, probably giving way of adhesions due to pressure of liberated gases when peroxid reached deeper parts of tract. From this time on marked aggravation of symptoms, increased discharge, great pain on movement, some fever, final and complete recovery about five months after operation.

CASE 4.—Chronic tubercular peritonitis with sacculation by formation of an artificial diaphragm across lower part of retracted intestines and mesentery. Operation for movable kidney a year earlier showed no tubercular disease in abdomen. History furnished by Dr S. P. Kramer, with whom this case was seen in second operation. Tubercular fistula, still discharging.

Mrs. E. T., housewife, aged 33. Mother of five children, all healthy. One brother dead from tuberculosis. Other family history negative. March 20, 1902, operated for floating kidney, right side. At that time pelvic organs normal. Present illness of about six months' duration. Abdominal distress, intestinal indigestion, great deal of flatus and pain in bowels. For past two months gradually increased dropsical swelling in lower part of abdomen. Temperature ranges from 99 F. in morning to 101 in evening. Examination shows fluid in abdomen, serous in character. Confined to lower segment of abdomen and pelvis. Abdominal walls tense, protruding; eversion of umbilicus, no tumor discoverable. Bowel resonance does not change when position of patient is changed. Abdominal walls thickened by peritoneal deposits. Ovaries and tubes matted together so that they can not be palpated. Menstruation regular and normal. Urine normal. Feb. 12, 1903, laparotomy. Ovaries and tubes markedly tubercular; parietal

peritoneum, having deposits of tubercles and tubercular hypertrophy. Pelvic organs separated from intestines by about three quarts of bloody serous fluid, which became partly gelatinous on exposure. Above the fluid there had formed a diaphragm of adhesion completely walling off the intestine from the cavity occupied by the fluid.

Ovaries and tubes removed. Abdomen closed without drainage. Primary union. On the twelfth day discharge from the lower angle of wound of about half pint of same fluid as before. At this point there formed a fistula the size of a pin head, which continued to discharge serous fluid up to date of discharge, March 14, 1903.

CASE 5.—Tubercular peritonitis wih slight ascites. Chronic pleurisy, operation, no improvement. Occurred in gynecologic service of my friend Dr. Withrow at Cincinnati Hospital.

E. Y., female, aged 32, housework, colored. Family history not known. Menstruation at 16, regular, lasting four to five days. Complains of pain in abdomen, especially low down in left side, extending down left leg. Some backache coming on in paroxysms; never pregnant. Admitted to Cincinnati Hospital Jan. 18, 1903. Unable to work for three months past. Swelling of abdomen began about three weeks before admission so severe that she could not wear her clothes. Swelling at first more marked on left side. Swelling varied from time to time. Not much pain. T. 100.2, P. 108, R. 24. Slight tendency to constipation. No appetite. Some resistance on palpation over lower half of abdomen, where resistance is more marked, where there is no tenderness on deep pressure. On day after admission dulness over left lower quadrant; remainder of abdomen tympanitic. Enema to unload bowel did not change physical signs. Next day dulness had completely disappeared. Vaginal examination negative. Evening temperature 102. Five days after admission dull area in lower left quadrant again present, extending somewhat beyond median line to right. Two days later dull area on left side does not reach to median line. I saw the case eleven days after admission. There could then be made out a small quantity of fluid in left chest, an indurated mass lying transversely across abdomen just below umbilicus, about 1½ inches in thickness, 4½ inches in length, not tender, slightly movable. Peculiar doughy elasticity of abdominal walls. Evening temperature reached 104. Laparotomy February 4. Incision 1½ inches in length in median line. Peritoneum thickened and gelatinous. About a quart of straw-colored fluid escaped. Peritoneum everywhere thickly studded with tubercles, those in the pelvis larger than on intestinal or parietal layers. Wound closed. Union by first intention. Apparently nothing gained by operation. Temperature ranges

for next month, during which time patient was under observation, continued same as before.

CASE 6.—Tubercular peritonitis, secondary to ulceration of intestine. Indurated bowel palpated through abdominal wall. Laparotomy; death in eight hours.

Mrs. E. D., aged 48. Married, one child, housewife. Still menstruating, never robust, but always well until one year ago. Began to have some pain in abdomen. Pain at first not constant; abdomen always sensitive, so that weight of hand accidentally resting on it as she lay in bed was painful. Not much appetite. Fever for three months with pretty constant diarrhea. Large liquid stools. Anorexia. Lately very profuse sweating. Abdomen tense, oval distension. A movable cylindrical mass felt in right iliac fossa. No pulmonary symptoms. Vaginal examination negative. Diagnosis, tuberculosis of bowel with peritonitis. Operation. Several areas of cylindrical induration of bowel discovered, making resection impossible. Pelvic viscera and entire parietal and visceral peritoneum covered with recent tubercles. Wound closed. Patient never rallied. Death in six hours.

CASE 7.—Secondary to tubercular pleurisy, adhesive form. Laparotomy. Death in three days.

J. H., saloonkeeper, aged 37. Brother died of phthisis. Always well until two years ago; had grippe. Pleurisy with exudation, relieved by repeated aspiration. Final obliteration of pleural sac. Abdominal distension gradually growing worse for six months. Slight fever, emaciation, obstinate constipation. Physical examination shows dulness over the base of the right lung posteriorly, due to the greatly thickened pleura. Breath sounds very distant. The abdomen distended, irregular areas of dulness in the midst of tympanitic resonance. Diagnosis, tubercular peritonitis with sacculation. Operation. All viscera matted together. A few small pockets of fluid are broken up; possibly eight ounces of turbid liquid escaped. Wound closed. Death on the third day.

CASE 8.—Pain in abdomen. No ascites, no fever.

Child, aged 11. Family history good. No previous illness. Sick four months. Characteristic distension of abdomen. Tenderness on pressure slight. Principal pain comes on after eating so that the boy fears to take food. Steady emaciation. No fever. Bowels constipated. In right iliac fossa a mass can be felt as though made up of adherent omentum; this is not particularly tender. Owing to prominence of gastric symptoms, test meal was given. Analysis showed normal condition of gastric juice. Treatment, rest in bed. Forced feeding. Still under observation.

FREQUENCY, AS DETERMINED BY AUTOPSY FINDINGS.

		Cases.	Autopsies.
Schalmack	(378)	54	5,425
Münsterman	(383)	46	2,837
Phillips	(384)	107	2,230
Sick	(521)	46	2,500
Steger	(395)	283	3,493
Adossides	(396)	74	4,815
Herringham	(586)	56	2,297
Borake	(394)	226	4,250
Total		792	27,847

Cases.	Males. Per cent.	Females. Per cent.	Males. Proport'n.	Females. Proport'n.
Under 10 years	45.5	54.5	7.0	9.0
10 to 20 years	14.3	85.7	1.0	6.0
Above 20 years	6.7	93.3	1.0	22.5
	(Lindner.)		(Dresduer.)	

	Males.	Females.
Adossides' table of children	34 per cent.	66 per cent.
Adossides' table of adults	10 per cent.	90 per cent.

	Cases.	Males.	Females.
Vierodt	24	19	5
Fenwick	46	30	16
Schalmack (in 5,425 autopsies)	54	33	21
Munsterman (2,837 autopsies, 903 tub.)	46	33	13
Phillips (2,230 autopsies)	107	89	18
Sick (2,500 autopsies)	46	31	15
Steger (3,493 autopsies, 929 tub.)	283	151	132
Adossides (4,815 autopsies)	74	53	21
Haene	46	18	28
Schnitz (all children under 13)	32	15	17
Schroeder	24	9	15
Total	782	481	301

Relative morbidity of male and female: Total, 782 cases; males, 481 cases; females, 301 cases.

DISCUSSION.

DR. E. J. BROWN, Decatur, Ill.—Dr. Fenger, several years ago, advised against operation in these cases, showing that the mortality with the so-called medical treatment was less than with the surgical treatment. I think it is very discreditable to the profession to give a bad prognosis in tuberculous peritonitis. I have seen three cases, in which the surgeon who was called in consultation advised operation, all improve without operation.

DR. JAMES TYSON, Philadelphia—As a physician my experience with tuberculous peritonitis has not been large, but so far as it has gone I have about concluded that to treat it medically is to temporize; in other words, that there is but one treatment that is likely to lead to satisfactory re ults, and that is surgical.

TUBERCULOSIS IN THE NEGRO.

SEALE HARRIS, M.D.
Health Officer of Bullock County from 1895 to 1903; President of the Southeastern Alabama Medical League; Councilor of the Alabama State Medical Association.
UNION SPRINGS, ALA.

Tuberculosis was a rare disease among the slaves in the southern states, yet, after a little more than a quarter of a century, it causes more deaths among the emancipated negroes than all the other infectious diseases. The negro death rate from tuberculosis is more than three times that of the whites from the same disease.

These statements, as startling to some as they are true, announce the motive which induced me to select for my theme on this occasion, "Tuberculosis in the Negro"; and if I can be the means of inciting any of you to consider the causes of this enormous mortality from that disease in the negro race and the measures to be taken for their relief, the objects of this paper will have been attained.

From the best information which I can gather, tuberculosis is unknown among the savage tribes in the interior of Africa. It is also unquestionably true that the negroes along the west coast of Africa were not affected by tuberculosis until the infection was carried there by the slave traders and colonists. However, since their exposure to the infection, the negroes in their native clime are not only more susceptible to tuberculosis than the white colonists who reside there, but the disease is more fatal among them. This is

shown by the following table (Dr. Rudolph Matas, "The Surgical Peculiarities of the Negro"), which gives the mortality from tuberculosis in the English colonies for the negroes and whites on a basis of each 1,000 deaths:

	English.	Negroes.
Jamaica	7.5	10.3
Dominica	8.3	16.8
Guiana	6.4	17.9
Ceylon	4.9	10.5
Gibraltar	5.3	43.0

Tuberculosis was so rare among the slaves in the southern states that some physicians contended that the negro was immune to tuberculosis. I have heard large slave owners, and several physicians who had large practices among the slaves in Georgia and Alabama, say that they never knew of a case of consumption among the negroes prior to the war. The disease was so rare that in 1830 a Cincinnati physician reported a case of a negro with "undoubted consumption," in which the autopsy showed "unmistakable evidences of the disease." There were no doubt a few cases among the slaves, but the disease was rare and confined almost entirely to the slaves in the cities, where they were overcrowded and the sanitary conditions were bad. The fact that there were so few cases of tuberculosis among the negroes while they were slaves was a great triumph of intelligent, though enforced, hygiene, and it also proves that tuberculosis is a preventable disease, because prior to the war it was not of infrequent occurrence among the whites of the South. That consumption is almost a scourge to the emancipated negro we all know, and the only reason why it was not so with the slaves was that their habits and sanitary surroundings were better than those of many of their masters.

Because of their indolence and improvidence, I am sure that the majority of negroes in the South do not get a sufficient amount of the proper nourishing food and the other necessities of life on which to maintain health and vitality up to the normal, and such lack of

nutrition, added to the worst possible ignorance and carelessness pertaining to personal hygiene, renders his tissues less resistant to bacterial infection of all kinds. In other words, it makes the tissues of the negro a suitable pabulum or soil for the invasion and propagation of the bacillus of tuberculosis. We also know that unless the tuberculous subject is well nourished he has but little hopes of recovery, and I am sure that one of the reasons why there are so few recoveries from tuberculosis in the negro race is that, as a rule, they are not provided with a sufficient quantity of palatable and nutritious food.

The following statistics of Charleston, S. C., which have been accurately kept for many years, show the increase in tuberculosis among the negroes since their emancipation: In 1860 the negro death rate was the same as for the whites, i. e., 12.00 per 1,000. The last census gives the white death rate in that city as 21.4, and the negro death rate of 43.1 per 1,000 of population. The deaths from "consumption," calculated at ten-year intervals, were in 1865, whites 57, negroes 74; 1875, whites, 54, negroes, 132; 1885, whites 57, negroes 209; and in 1895, whites 39, negroes 194. Estimated population in 1895 was, whites, 28,870; negroes, 36,295.

The census returns for the year 1900 show, in the "registration area," in which the statistics may be regarded as fairly accurate, that for every 100,000 of population there died from "consumption" 126.5 native whites and 485.0 of negroes.

I regret that I am unable to give any statistics regarding the mortality from tuberculosis in Alabama and the other southern states. I studied the census returns carefully and found but little that I thought would be of interest or value. The following letter came to my relief, and caused me to destroy most of the data which I had compiled from the census report, and I quote from it to show the fallacy of deductions based on mortality statistics of the United States Census Report, except those for the "registration area":

CENSUS OFFICE, WASHINGTON, D. C., Sept. 20, 1902.

Dr. Seale Harris, Union Springs, Ala.

Dear Sir:—I regret to state that this office can not furnish you any data that are of any value concerning either the general death-rate of the white and colored in the United States as a whole, the southern states, or for South Carolina and Alabama individually, or the mortality of these classes from tuberculosis.

The return of deaths in all of the southern states and in most of the other states was secured by inquiry of the enumerators, and falls very far short of being complete. In fact, the return is absolutely worthless for the purpose of computing death rates and any conclusions based on them would be erroneous. It is only in those states and cities that have efficient systems of registering deaths that we are able to secure anything like a correct return of the deaths that actually occur which can be utilized for determining the death rates. Unfortunately, there are no southern states, and very few cities which have a complete system of registration, and prior to 1890 this source of information was not utilized in any way in census statistics. Very respectfully,

W. A. KING, Chief Statistician.

The statistics for some of the southern cities are regarded as being as accurate as those for the registration area, and below I give reports of five representative southern cities:

RATES PER 1,000 OF POPULATION.

	All Causes.				Tubercular Diseases.			
	1890.		1900.		1890.		1900.	
	Wh.	N.	Wh.	N.	Wh.	N.	Wh.	N.
New Orleans	24.8	39.4	20.7	39.7	2.50	5.87	2.38	6.13
Mobile, Ala.	21.5	34.5	21.0	32.5	3.04	6.08	3.08	6.09
Charleston, S. C.	21.4	42.2	21.4	43.1	3.55	6.86	1.65	6.21
Savannah, Ga.	24.7	37.8	22.6	42.2	3.71	5.44	2.64	4.91
Richmond, Va.	22.3	37.6	18.1	32.8	2.30	4.11	2.05	3.94

The statistics which I have collected for Union Springs, with an equal population of whites and negroes, and which I think are reliable, show that in the past eight years there have died from tuberculosis fourteen whites and thirty-nine blacks; and of the considerable number of negro deaths reported with the cause of death unknown, I am sure that several died of tuberculosis. I do not know of any other statistics of any value for the small towns or rural districts of the South,

for the reasons that the majority of deaths occur without the attendance of a physician, and are reported by midwives and undertakers, who know nothing of the causes of death.

The great prevalence of tuberculosis is not so much due to any inherent tendency to the disease as to their habits and environments, which render their tissues less resistant to the tubercle bacilli, and for the same reasons they fall easy victims to its ravages. However, it is no doubt true that there is a less development of lung tissue and the accessory muscles of respiration among the negroes than for the whites. Dr. McDowell, a surgeon in the United States Army, made frequent autopsies on white and colored troops during the Civil War, from 1861 to 1865. He found that their chest measurements were full (up to the army standard), but their lungs were always lighter and smaller than those contained in the same sized chest among the white troops. The statistics of Otis and Woodward for the same period are interesting. They found a less lung capacity and smaller brain for the negro troops than for the whites. They also found that of whites who contracted tuberculosis during the war, one out of two and one-half died, while of negroes twelve out of thirteen died. Dr. Russell, another army surgeon, states that the average weight of the negro was four ounces less than that of the white.

F. L. Hoffman[1] gives the following interesting table, which he compiled from the report of the Provost-General (Vol. I), and the reports of the Surgeon-General, U. S. A., 1893-94-95, which shows the difference in chest expansion of the slaves who entered the army, and of the emancipated negro of thirty years later.

This table clearly proves that there has been a diminution in the chest expansion of the negro since his release from slavery.

1. Race Traits and Tendencies of the American Negro, page 161.

COMPARATIVE CHEST MOBILITY OF U. S. RECRUITS, 1861-65 AND 1892-94:

Age Periods.	U. S. Army, 1861-65. White.	U. S. Army, 1861-65. Colored.	U. S. Army, 1892-94. White.	U. S. Army, 1892-94. Colored.
Under 20 years.....	3.26	3.17*	2.82	2.56*
20 to 24 years.....	3.25	3.28†	2.86	2.52*
25 to 29 years.....	3.22	3.25†	2.93	2.62*
30 to 34 years.....	3.22	3.19*	2.96	2.64*
35 to 39 years.....	3.26	3.18*	2.94	2.57*
40 to 49 years.....	3.18	3.34†	2.84	2.52*
All ages	3.24	3.23*	2.93	2.58*

* Excess in favor of white. † Excess in favor of colored.

Gould has proven that the average lung capacity of the negro is considerably less than that of the white. Thus, for the average height of 5 feet 8 inches, the pulmonary capacity was for the whites 188.5 cubic inches, for the negro, 167.5 cubic inches—giving a mean difference in favor of the whites of 15.0 cubic inches. The pulmonary capacity of the average chest measuring thirty-six inches in circumference was for the whites 186.5, for the negro, 170.5—thus showing a mean difference of 16 cubic inches in favor of the whites.[2] Dr. R. M. Cunningham[3] has called attention to the "more or less encroachment of the abdominal organs on the thoracic viscera, and for that reason the perpendicular diameter of the chest of the negro is less than that of the same sized white." My observations, carried out on the line of Dr. Cunningham's ideas, from the examination of many healthy chests among the negroes, show that the normal liver dulness on the right side, sometimes extending as high as the fifth rib, and the tympanitic note over the stomach on the left side (Traube's semilunar space) of the negro are higher than that of the whites. I have also noted that Litten's diaphragm phenomenon is less marked in the normal negro than in the white.

Thus all observers agree that there is less development of the lungs of the negro than for the whites, and this physiologic difference no doubt accounts for, in a measure, the inordinate mortality from consumption,

2. Gould, Military Statistics, pp. 480 and 493.

3. The Negro as a Convict, Transactions of the Alabama State Medical Association, 1893, p. 321.

pneumonia, and the other pulmonary diseases among the negroes.

Dr. M. L. Perry, pathologist of the State Sanitarium of Georgia, examined the brains of 100 whites and the brains of 100 negroes. He found that the average weight of the brain of the negro was 100 grams, or 3½ ounces less than that of the whites. It is also well known that in the brain of the negro the convolutions are less complex, and the sulci are shallower than that of the Caucasian, which, added to the fact that his brain is smaller, gives the negro considerable less gray matter than for the white. Thus the negro's brain approaches nearer the Simian type than that of the Caucasian, which, according to our ideas of evolution, would place him on a slightly lower scale as a man than his white brother. Probably for this reason they are lacking in the inherent vital nerve forces which in serious illness is sufficient to make them succumb to all diseases, particularly those of a wasting nature like tuberculosis, than does his white brother, who is endowed with a better developed brain structure. I am sure that even under the most favorable circumstances, which I am sorry to say is given to but few of their sick, the negro dies more readily from all forms of disease. I have known of many whites who have recovered from consumption when the treatment was begun early, but of the large number of cases which I have treated among the negroes I have seen but a few who showed even temporary improvement, and only one who recovered from the disease.

I would not be understood as meaning that there are any inherent differences in structure of the tissues of the whites and blacks—only a difference in development. The whites have many centuries of enlightenment and civilization under such varied conditions that there has been the psychical and physical development, which fits him to endure the manifold dangers of disease which beset us in our struggle for existence in this complex

age, while the negro, a century or two ago, was a savage, perhaps a cannibal, who needed only to provide himself with food, of which nature so lavishes both animal and vegetable in the forest of the tropics of Africa. The heat of that climate made it unnecessary for him to have clothing to protect his body, and with the low altitude and the warm, humid atmosphere, less oxygen was required to maintain body temperature, so there was a corresponding lack of development of the lungs of the native African. This, the hygienic life which he was compelled to lead as a slave, was overcome to a great extent, as is shown by the table compiled by Hoffman, which makes it clear that the chest expansion of the negro was nearly, if not quite, equal to that of the whites in 1861-5, while in 1892-4 it was considerably less. However, the life of a slave did not fit the negro to provide for himself in a climate entirely different in environment to that which his ancestors had lived for ages, and the utter incapacity of the majority of negroes to take care of themselves and families in this climate is the great underlying reason why tuberculosis, which finds its victims in those of a lowered state of vitality, has increased so rapidly, and almost always proves fatal among the members of this unfortunate race.

The filthy habit of spitting shows its highest development among the colored population. Nearly all the men chew tobacco and their women use snuff, and the healthy and the tuberculous alike spit on the floors of many of their homes and churches. The best of their churches are provided with boxes filled with dry sand or sawdust into which they expectorate. The sputum, either on the floors or in the boxes, becomes dry and gets into the dust of the atmosphere, and the bacilli are inhaled in large numbers, and, lodging on lung tissue already weakened from the causes of which I have spoken, they find a favorable soil for their development. When we remember that the consumptive, with a cavity in his lungs, expectorates from 2,500,000,000 to 4,000,000,000

germs in twenty-four hours (Nuttall), and that tuberculosis is so prevalent among the negroes, we can see that this is no chimerical idea that their churches are among the great disseminators of tuberculosis.

Syphilis is not in the least allied to tuberculosis, but it is a well-known fact that syphilitics die from tuberculous diseases. Syphilis is certainly one of the great predisposing causes of tuberculosis, and the great increase in syphilis in the past thirty years has been *pari passu* with that of tuberculosis.

Gonorrhea, on account of their habits of licentiousness, is one of the most frequent diseases among negro men, and every physician who has examined many negro women knows how frequently they are afflicted with "pus tubes." Osler says: "Tuberculous peritonitis in America is a more common disease in the negro than in the white race, and recently collected statistics show that females predominate." Kelly has shown that a very considerable proportion of all cases is secondary to tuberculosis of the fallopian tubes. It is also well known that in man tuberculosis of the seminal vesicles often precedes the development of the disease in the peritoneum. My own observation has been that tuberculous peritonitis is a very much more frequent disease in the negro than in the white race, and that it is several times more frequent in women than in men. For this reason I am inclined to the opinion that gonorrheal salpingitis, prostatitis and seminal vesiculitis prepare the soil for the bacilli of tuberculosis, and from these loci minoris resistentiæ the disease originates and extends to the peritoneum. Therefore, it seems that the gonococcus is the indirect cause of a considerable proportion of negro deaths from tuberculosis.

A great many negro children die from tuberculous peritonitis, which is frequently secondary to the disease in the intestines, and I have no doubt but that the causes of death in many cases reported as gastroenteritis are really enteritis and peritonitis of tuberculous origin.

Nasal catarrhs, tonsillitis, bronchitis and other troubles which are commonly called colds, which are frequent precursors of phthisis pulmonalis, are very prevalent among the negroes, and are rarely ever treated. They are exposed to all kinds of weather, and in winter many of them do not have sufficient clothing to keep the surface of the body warm. Their lack of personal cleanliness, as is demonstrated by their proverbially characteristic odor, which, by the way, is not so noticeable in those who bathe frequently and regularly, prevents the skin from acting freely, therefore, predisposing factors in the development of so much consumption among the negroes, and for the same reasons they are prone to the most virulent types, i. e., acute pneumonia phthisis, phthisis florida or "galloping consumption." Those of us who have treated many cases of tuberculosis among the negroes have seen them die from the acute pneumonic type in a few days or weeks. I am quite sure that many of the deaths reported as due to pneumonia really result from acute pneumonic or broncho-pneumonic phthisis.

While the lungs of the negro are the organs most frequently attacked by tuberculosis, he is subject to all its protean forms. Tuberculous pleurisy and meningitis and tuberculous disease of bones and joints are of frequent occurrence among them. Indeed, there is hardly a tuberculous condition which I have seen described that I have not seen in the negro. Lupus seems to be less prevalent among them than any other form of tuberculosis. Lupus, however, seems to be a rare disease in the South.

That tuberculosis is so prevalent among the negroes everyone admits, yet every year goes by and almost nothing is done to stay the hand of this fearful disease, which, with the other diseases to which the emancipated negro is especially prone, threatens the extinction of the negro race and, indeed, greatly endangers the happiness and lives of the whites of the South. This is no new

theory with me. Hundreds of other physicians in the South hold to the same opinion, but the whites generally and the negroes do not seem to realize the danger which threatens to almost exterminate the "ward of the nation."

F. L. Hoffman[4] says: "It can be proven at the present time that the colored race is subject to an inordinate mortality from consumption and respiratory diseases, which will menace the very existence of the race in the not far distant future."

Dr. Cunningham says in his paper on the "Negro as Convict": "In conclusion, I will submit that the race problem will be largely settled in the event of public works being established throughout the South, which means ready money for the negro; that means the profitable practice of prostitution; that means gonorrhea and consequent sterility; tuberculosis will do the rest."

Dr. Rudolph Matas,[5] in his splendid and comprehensive paper on "The Surgical Peculiarities of the American Negro," makes the following statement: "Tuberculosis has been called the "great white plague"; but if we look at this matter statistically and through the eyes of all experienced observers we would with greater propriety call it the black plague, as it is unquestionably the dominant cause of the increasing death rate of the negro in this country."

Dr. P. B. Barringer, Chairman of the Faculty of the University of Virginia, in his masterly address before the Montgomery (Ala.) "Race Congress," placed tuberculosis as the principal factor in "that grand but ghastly tragedy—the sacrifice of a race."

I am not an alarmist, and this paper, which calls particular attention to the weak points of the negro, is written for no other purpose than to call attention to

4. Race Traits and Tendencies of the American Negro, p. 82.
5. Transactions of the American Surgical Association, vol. 14, p. 523.

the alarming increase of tuberculosis in that race, with the hope that something may be done which will lessen the frightful mortality of the disease in this generally kind-hearted people. The South is doing a noble part by the negro in the line of education, and I am glad that it is so, because without the guiding and protecting hand of a master, the African and his descendants in this climate and this strenuous age can only hope to survive the struggle for an existence through the education of their minds and the training and development of their bodies. If for no other reason than that of self-protection, it is high time that the whites of the South, on whom must fall all the cares and responsibilities of managing the affairs of state for the public good, should take steps to prevent the continued spread of tuberculosis among the negroes. If our cooks, nurses and other servants have the disease there is danger of our families becoming infected. Incidentally, I will mention that I do not believe that tuberculosis is decreasing among the whites of the South, and whatever steps are taken to lessen the prevalence of the disease should apply equally to the whites and negroes.

The methods to pursue in limiting an infectious disease like tuberculosis should be directed toward (*a*) increasing the strength and vitality of the individuals of the race, thereby rendering their tissues an unsuitable soil for the invasion and propagation of the specific germs of the disease, and in (*b*) preventing his exposure to the infective agent, as by the isolation of those already afflicted, and by destroying the organisms causing the disease.

The first proposition presented is the development and upbuilding of the minds, morals and bodies of a "child race," an alien people, placed under conditions and environments which would seem to be unfavorable to the growth and stability of the race, and wholly unlike those under which his ancestors had lived for centuries. In a paper like this I can only discuss this phase of the

question briefly, but those who are interested I would strongly urge to read "The Race Tendencies and Traits of the American Negro," by F. L. Hoffman, the able statistician of the Prudential Insurance Co. of America. He has collected and published in this book, at an enormous expenditure of time and energy, the largest and most valuable statistics bearing on all phases of the negro question that I know of in all the literature on the subject. He has applied statistical methods in forming his deductions, and, being a German by birth, and, therefore, free from the so-called race prejudice, his conclusions are of great value, and the facts and figures which he has presented can hardly be questioned.

Much may be done to develop the negro through education applied on the right lines. He has every opportunity in the South that the whites have of obtaining both a common-school and collegiate education, but the moral, physical and hygienic education of the race is almost wholly neglected. By educating the children in these matters they would instruct their parents on the hygiene of their persons and their homes. Their school buildings should be inspected by the health officers in every county and should be kept in a sanitary condition. Regular physical exercises, directed especially toward developing their lungs and the accessory muscles of respiration, should be a part of the daily routine of school duties. The children should be taught the importance of taking frequent and regular baths; that filth means disease, and cleanliness should be required of them. They should be shown the direful effects of immorality on their bodies as well as their souls. A higher standard of morals and education in their ministry and among their teachers would help much in this direction.

Regular daily lessons and talks on practical hygiene should be a part of the curriculum of every school. They should be taught how to live in their homes; that there "cleanliness is next to godliness"; that fresh air and sunlight are among the great boons to humanity, and

if they would have health they must have plenty of both in their homes. They should be shown the dangers of overcrowding; that health can not be maintained when more than two or three sleep in the average-sized room; that there is danger in sleeping in the room with any sick person, especially one with a cough. They should be doubly impressed with the danger of spitting on the floors, walls, or any place where the sputum can dry and dust particles get into the atmosphere; that the germs of consumption, which disease, the youngest of them know, is the cause of many deaths of their relatives and friends, is carried through the atmosphere and breathed into their lungs, and that the dried sputum is practically the only source of infection. They should be instructed in the hygienic management of a case of tuberculosis; that there is danger in even a casual association with a consumptive who is careless in his habits of spitting, but that there is little danger of contracting the disease even by close association with a person who has consumption, and who is cleanly in his person and at his home, and who is careful to expectorate into vessels containing dilute solutions of an antiseptic fluid, or into spit cups, which are burned before the sputum has time to dry.

They should also be taught that health and happiness come from regularity of habits and work, diet, recreation and sleep, and that a liberal quantity of all are required to keep in perfect health. Above all, they should be taught the manliness and honor of physical labor, and that their education fits them for their life's work, and not for a life of ease and luxury; that there is as much honor in farming as in teaching or preaching. Industrial education is, therefore, a movement capable of doing great good for the race. They should be taught to economize, to save their money and put it in homes and farms. That for their race the dangers of Sodom lie in the towns and cities; that there lurk consumption and death.

These things, and much more, should be taught to the negroes, not only for the sake of right, but to make them stronger physically, so that they may resist the infection of tuberculosis.

The isolation of those infected with tuberculosis would unquestionably limit the disease in a great measure, but the difficulties of enforcing or carrying into effect in any way the isolation of the thousands of indigent tuberculous negroes in every state in the South are so great as to make the suggestion impracticable, though something may be done on that line.

The state could enforce the isolation of the tuberculous patients in their convict camps and penitentiaries, which are often great harbingers of the infection of tuberculosis.

The establishment of sanatoria for the isolation of the tuberculous should receive more attention from philanthropists and the civil authorities. In Massachusetts and other states they have been successfully conducted by the state for several years. Such establishments need not call for expensive buildings, but rather the cottage system, or even on the plans of the plantation quarters for slaves, where the negroes enjoyed such immunity to tuberculosis. They should be located in the country, where the inmates can get plenty of outdoor exercise and when able do farm work, and assist in paying the expenses of such institutions. Very few negroes would be able to pay their expenses at these sanatoria, and they would necessarily have to be charitable institutions, but they could be maintained at a probably less per capita expense than those at the hospitals for the colored insane, and I am sure that the same number of tuberculous negroes are a source of greater danger to their families and to their friends than the same number of their insane, if left in the care of their families. Aside from the isolation feature of sanatorium treatment for consumptives, the hygienic education that it gives to the patient and through him to

his relatives and friends will materially aid in protecting many who are exposed to the disease.

There should be several such sanatoria in every state. Even if they should entail a great expense to the state, in the end it will be economy, because in a few years, perhaps a quarter of a century, when tuberculosis will have a chance at the weakened offspring of this and the coming generation of negroes, the havoc will be so great among that race and the danger to the whites so clear that enormous expenditures will have to be made in order to relieve the acuteness of the situation. The state and municipal authorities will spend thousands to prevent an epidemic of smallpox or yellow fever, yet spend almost nothing to prevent the spread of a disease which destroys a hundred times as many of our citizens, and which entails far greater and more prolonged suffering than both of those diseases, besides the direful effects on the offspring of its unfortunate victims.

Unquestionably, tuberculosis should be managed by the health authorities, as are the other infectious diseases. In some of the cities every case of tuberculosis is required to be reported to the health officers, who instruct the patient and his family as to the methods to use to prevent the spread of the disease. The patient is not interfered with in any way, except that he is expected to employ the usual methods of destroying the sputa, and to follow the simple rules of hygiene to protect those with whom he comes in contact. After the removal or death of the patient the patient's room, or house, is disinfected just as for other infectious diseases.

The health officer of every city and county should be required to understand the use of a microscope, and it should be a part of his duty to examine, without charge, every specimen of sputum sent to him. He should also be skilled in physical diagnosis, so that the physician or patient could call him in to clear up the diagnosis in cases of doubt, as is required of him in the other in-

fectious diseases. We all know that tuberculosis is one of the curable diseases if the diagnosis is made early and intelligent treatment is applied. If the public could be educated to call in the health officer in the early stages of tuberculosis and thereby become certain of the diagnosis, not only would many of the patients be saved, but it would aid in lessening the infection, because it would give the health officer the opportunity of instructing the patient and his family on the hygienic measures necessary to prevent the spread of the disease before the home had become infected.

Since the tuberculous patient is dangerous to his friends and the community only from his ejected sputa and directly in proportion to his habits of spitting, the antispitting ordinances, which are already in force in all cities for their public buildings, cars and streets, are doing great good by lessening the chances of breathing the infection from the atmosphere. The states should adopt such ordinances for public buildings of all kinds, including schools, churches and depots. Of course, they would not always be enforced, but a placard announcing the ordinances and stating the reason for their adoption, would call attention to the dangers of spitting, and would aid in molding public opinion against the spitting evil.

Physicians who come in contact with negroes at their homes should, more than any other class of whites, consider it a duty to instruct them in the simple methods of hygiene, particularly when one of the family is afflicted with tuberculosis. The whites should be educated along the same lines, and when the laity, both among the whites and blacks, properly understand the nature of tuberculosis, the life history of its germs, and the methods to employ to prevent its spread, then, and not until then, may we hope to limit the ravages of this disease, the greatest enemy to mankind, and which is the principal agent that threatens the extinction of our "brother in black."

SHOULD THE TUBERCULOUS PATIENT KNOW THE TRUTH REGARDING HIS CONDITION?

C. P. AMBLER, M.D.
ASHEVILLE, N. C.

All physicians who practice at health resorts frequented by patients suffering from pulmonary tuberculosis find that comparatively few of these patients have any conception of their real condition, and in the rarest instances have they been carefully instructed as to the necessities for recovery.

The profession generally seems to be of the opinion that tuberculous patients should not be told the nature of their trouble, but the average tuberculous patient generally finds out the true nature of his disease, and when he does he loses faith in his physicians, and frequently, very justly, will censure them for not telling him the truth.

How is it possible to get the coöperation of the patient; to have him use care in the disposal of expectoration; to avoid violence and over-exertion in exercise, and many other things so important in the detail management of the case, if he does not even know that he has a serious trouble with which to deal?

The truth may depress him; probably it will for a short time, but it will not do him near the harm that his ignorance will result in, if he does not appreciate his condition. Every state and municipality is awaking to the danger of infection from the dried sputum of tuberculous patients. Our journals are full of papers advo-

cating antispitting ordinances, institutions for the tuberculous poor and other safeguards intended to protect the public. But why, pray, should we have ordinances and legislation unless the patient is always to know the truth? Legislation we need, but we first need more candor on the part of the physicians.

Formerly a diagnosis was only made in the advanced cases. The patients all promptly died. Result: the laity watched these cases and observed, "they all die." But little idea was ever entertained in those days of the danger to others. We, as physicians, by our tardy diagnosis, have educated the public to the belief that the disease is uniformly fatal.

It is the duty of the physician to explain to the patient the dangers to himself and to others; to point out where he is most apt to make mistakes; to give explicit directions regarding his hygienic life, including diet, exercise, work, physical or mental application, secretion and excretion, sexual relation, and in fact, to go fully into the whole question of the hygienic life. Such instruction on the part of the physician is much more important than the prescribing of drugs with an idea of "cure" in this disease. These instructions and directions should apply to the incipient as well as the advanced cases. To get the coöperation of an incipient case (and his coöperation is of the most vital importance) he must know the truth.

The patient should thoroughly understand the possibilities of communicating the disease to others, and also that he can infect sound tissues in his own lung if he is not careful in following directions for disposal of expectoration. How can we expect care on his part if he does not understand the danger, both to himself and his friends? Many will use care on account of their friends, and others more selfish can only be appealed to with good effect when they realize that they endanger themselves.

The laws of health demand that the patient fully realize the danger to self and others.

Appeal to the patient on the ground that it is unkind to his friends not to make every effort to protect them. If this plan had been followed more in the past the disease would not be so common to-day.

If tuberculosis produced the rapid disfigurement and distress that many of our acute diseases do, or was the active developing disease that smallpox is, we would have quarantined against it long ago. Its slow development makes it hard for the laity to realize that danger exists. Quarantine is unnecessary in tuberculosis, provided the patient is properly instructed and these instructions carried out. The whole country is awaking to the fact that something must be done. We, as physicians, should realize that one-sixth of our death rate each year is caused by tuberculosis; in other words, one-sixth of those who die this coming year in this country will die of this disease. Those who die from tuberculosis the two years following this, in the great majority, already have the disease in an incipient form. It is to their interest to know the truth to-day. It is to the interest of the general public that they know it; that they are fully instructed and cautioned. They consult us for this purpose; it becomes our duty to determine their condition early and so inform them. We have a duty to the other members of a family as well as the patient. Can the state house this great army of patients? Can we quarantine them if we would? No. How can we, as physicians, prohibit the infection of others? By educating the people to their danger and instructing the patient as to his duty.

Is there any way in which the necessary hygienic measures can be secured without the coöperation of the patient? Can we ever get his coöperation unless we tell him that he has a tuberculous disease?

The patient comes to us for the truth. It is our duty to show him that the disease is not the fatal disease that

the public have imagined, if instructions be followed. Every physician should keep at hand printed matter, instructions or directions, to put into the hands of the tuberculous patients.

We have for years told our patients that this disease is like unto a fire in a house. If you get after the fire when the match is struck it is easy to put out; let it burn until one whole room is on fire and you have a much more difficult undertaking; let it burn until the house is on fire from garret to cellar and it is sure to burn up.

For the protection of our neighbors, as well as our own property, we all handle fire with care. If a friend's house is on fire would you tell him? A thousand times more important is it to tell him when we find that he is infected with tuberculosis; here the greatest possession he has on earth is at stake. The sooner he knows it the better.

By telling the patient the truth, we may occasionally shorten the days of a few, but in the long run will save the lives of many hundred by following this policy. Our surgeons operate to save life, even when grave danger exists by such an operation to the patient. If our tuberculous patients are not told the truth, the danger is not only to the patient, but to all his friends included.

It is an unpleasant duty to perform, but one from which no physician should shrink; and especially should patients never be sent away from home without knowing their true condition.

How frequently are we consulted by some poor soul who had left home and arrived at the resort suffering, as he says, from a "slight throat trouble"; arrived may be in the beginning, and, perhaps, well advanced in pulmonary or laryngeal tuberculosis; without money, expecting to procure work to pay for his board and keep; arriving under the fond delusion that by climatic influence his trouble will soon disappear; with no conception of the necessities for recovery; no knowledge

of the gravity of his disease; in many instances his chances were a thousandfold better at home among his friends, had he been told the truth, given explicit instructions, given less medicine and more time by his physician.

The tuberculosis patient who does not know his true condition rarely improves. As his case progresses he becomes discouraged and loses confidence in his medical advisers, thinking they do not understand his case. Can he be blamed?

On the other hand, the patient who is told the truth may temporarily be depressed, but he quickly responds and enters heart and soul into the fight before him. Rarely indeed do we find a patient who has been properly instructed and handled (without misrepresentation) who does not look forward to recovery. Phthisical patients are, as a rule, hopeful. Not so the patient who does not know what is the matter.

Intelligent pulmonary patients sent away from home without being told the truth frequently, from observation alone, find out their condition. When they make this discovery themselves it creates distrust of the physician at home and places the physician at the resort in an embarrassing position, where the truth from him now will reflect on the physician who may have referred the patient to him; or, he likewise must misrepresent the facts to the patient with the result that later he will be censured, with the home physician, for "not finding out what the trouble was."

True, there are exceptional cases where the patient is accompanied by the parent or some one who can control him, when the whole truth need not be given to the patient, but these cases are extremely rare. We have in the past relied too much upon drugs; we have been too free with our prescriptions and too careless in our instructions. If we would give the patients more of our time and less of our dope it would be better for the patient. The good results obtained from

institution treatment of this disease are dependent, in a great measure, on the fact that the patients know their condition and are under constant supervision and instruction.

For the good of our tuberculous patients; for the protection of their friends and the public generally; for the protection of ourselves, both physically and from censure, let us fully inform our tuberculous patients as to their disease, and when so informed take the time to properly instruct them what to do and what to avoid.

TYPHOID FEVER.

SOME UNCONSIDERED HINDRANCES IN ITS PROPHYLAXIS.

JOHN S. FULTON, M.D.
BALTIMORE, MD.

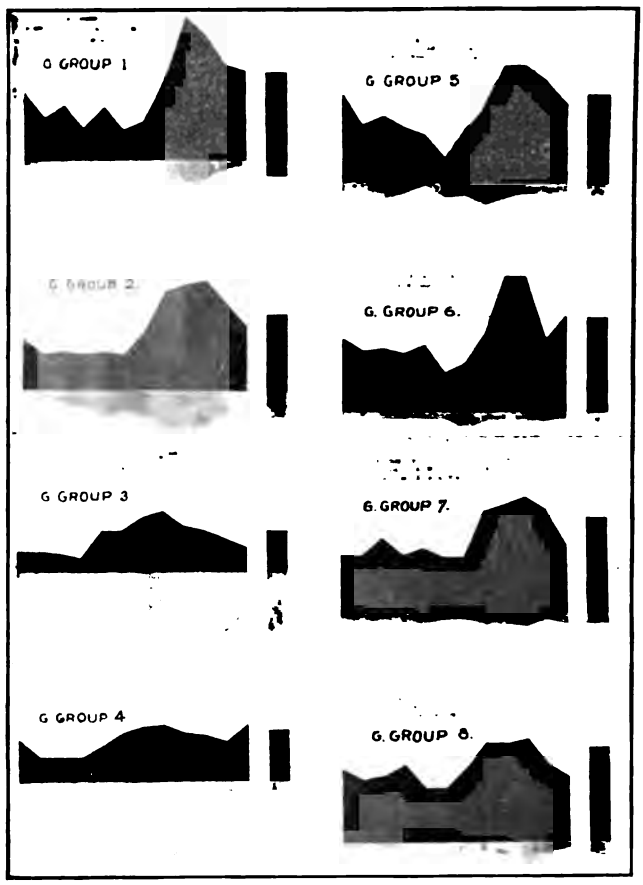
For fifty years we have known that drinking water is the chief vehicle of typhoid infection, and this knowledge has enabled great cities to make measurably effective defense against typhoid fever. For more than twenty years it has been known that the whole problem of preventing typhoid fever lay in the disposal of typhoid dejecta. Knowing so much, it would seem that typhoid fever might have long since been reduced to insignificance as a cause of death. But the bad eminence of the disease is as conspicuous to-day as it was twenty years ago.

In the grewsome story of typhoid among the United States troops during the Spanish-American War, we were confronted by a fact so humiliating that the profession has not yet had courage to consider fully its great significance. We learned that of every one hundred cases of typhoid fever sent to division hospitals, one-half had escaped the diagnosis of regimental surgeons. This enormous defect of professional skill was exposed, not by typhoid in its rarer and more subtle disguises, nor by the very mild infections, but by plain, uncomplicated typhoid fever, which puts a man to bed for three to six weeks; such typhoid as all physicians see year after year and recognize five or six times in ten. Restrictive measures can avail us little if the enemy can maintain effective disguise while fully armed and accoutred. The failure

of definite and effective means to restrict the prevalence of typhoid fever is chargeable primarily against American medical schools, which do not teach young men to recognize the commonest continued fever of the United States.

Before the fight against typhoid fever can become hopeful one barrier must be leveled: a ponderous inertia which American medical education has imposed on the medical mind in respect to one disease. Until we are relieved of the dead weight of authority, the thraldom of the text-book, we shall go on misinterpreting, in 40 per cent. of instances, the clinical phenomena of typhoid infection. While dogmatic teaching is rapidly giving way to natural methods of study, there is in the portfolio of many a professor of medicine one lecture, or a set of lectures, which will be given up with great reluctance. The subject is typhoid fever, and the vivid descriptions so impress the students that they ever after require each case of typhoid to be as good as the description, to wear all its insignia, and to proclaim aloud its full name with decorations and titles. The prophylaxis of typhoid fever would gain an impetus from the conflagration of all such literature. Typhoid fever is taught too much; it is studied far too little. This defect of medical education has no preferred habitat within the confines of America. The glaring delinquency of Army medical men owed little to the latitudes in which it was demonstrated. These observers came from all parts of the land, and brought with them such light as their previous training and professional associations afforded. The men who entered the Army medical service in the late war were not more delinquent than those who remained in civil practice. There is a broad humor in Carroll's story of a solitary regimental surgeon who had no malaria among his fifty fever patients. He had a foreign accent, and had not acquired the American delusion that malaria is a considerable contributor to the mortality of the country.

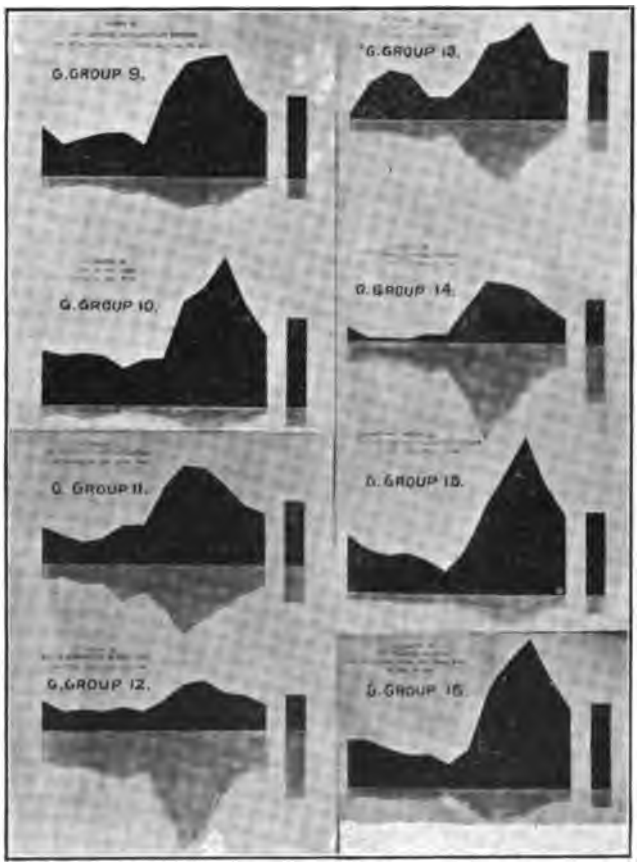
In 1898 the Special Commission on Typhoid in the United States Army Encampments testified that no fatal malarial infections occurred to the soldiers in any part of the United States. In 1900 the profession of this



Section 1 of Chart 1.

country testified through the Census Bureau that for every seven persons who died of typhoid fever, three others died of malaria. In the south Mississippi belt the testimony is that twice as many deaths occurred from

malaria as from typhoid. Among the twenty-one geographical divisions of the country this one, the south Mississippi belt, showed the largest error in numbers, but a worse error was that of the central Appalachian

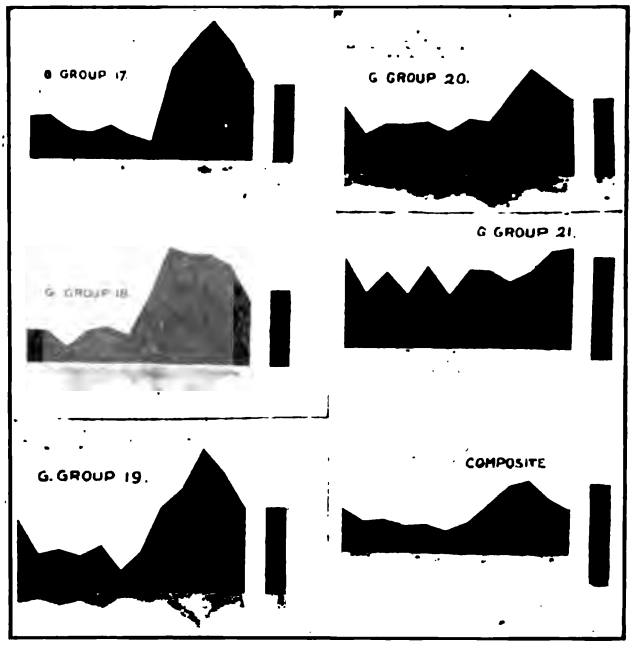


Sectoin 2 of Chart 1.

region, where malaria is not even a considerable cause of disability, but where, nevertheless, it was charged with one death for every ten charged to typhoid.

I have plotted for each of the twenty-one grand groups

the mortality returns of both typhoid and malaria by months, and the seasonal curve itself points a suspicion that confusion between the two diseases is everywhere more or less prevalent. Indeed, the charts show the malaria curve below obedient to the typhoid curve above as faithfully as the shores are reflected by the surface of a lake. The seasonal apex for the two diseases



Section 3 of Chart 1.

Chart 1 shows the reported mortality from typhoid fever and malaria in 1900 according to United States Census, by months and by Grand Groups. The black spaces indicate typhoid and the shaded spaces below indicate malaria.

coincides in ten of the twenty-one groups, five times in August, once in September, four times in October. In eight groups the apex of the reported malaria mortality anticipates the typhoid apex by one month. In the whole Appalachian region, which runs from northern New York to within 200 miles of the Gulf coast of Ala-

bama, the malaria is a midsummer madness, the apex being reached in July.*

In the mid-Atlantic coast region, which includes parts of New York, New Jersey, Delaware, Maryland and Virginia and the whole of the District of Columbia, the Census figures show the typhoid fever and malaria mortalities in a ratio of 72 to 28. The Delaware State Board of Health recently made an extended study of the subject by modern methods, and concluded that practically all the remittent and continued malarial fevers were in fact typhoid. None of them was malarial. In Maryland, where examinations of blood for the malarial parasite have been made for four years, the parasite has been found in but 5 per cent. of all the specimens submitted. My personal belief is that fatal malarial intoxication does not happen to one citizen a year in Maryland, though malaria is in some parts of the state a considerable cause of disability.

Of all the mortality charged in the south to malaria, half, perhaps, is really due to typhoid fever. The United States Army's experience would indicate that much more than half is due in fact to typhoid, and certainly more than 50 per cent. of the mortality so charged is not due

---

* These Grand Groups do not respect state boundaries, but physical characteristics, as may be understood from their designations as follows:

Grand Group 1. North Atlantic Coast Region.
Grand Group 2. Middle Atlantic Coast Region.
Grand Group 3. South Atlantic Coast Region.
Grand Group 4. Gulf Coast Region.
Grand Group 5. Northern Hills and Plateaus.
Grand Group 6. Central Appalachian Region.
Grand Group 7. The Great Northern Lakes.
Grand Group 8. Interior Plateau.
Grand Group 9. South Central Appalachian Region.
Grand Group 10. Ohio River Belt.
Grand Group 11. South Interior Plateau.
Grand Group 12. South Mississippi River Belt.
Grand Group 13. North Mississippi River Belt.
Grand Group 14. Southwest Central Region.
Grand Group 15. Central Plains and Prairies.
Grand Group 16. The Prairie Region.
Grand Group 17. Missouri River Belt.
Grand Group 18. Western Plains.
Grand Group 19. Heavily Timbered Region of the Northwest.
Grand Group 20. Cordilleran Region.
Grand Group 21. Pacific Coast Region.

to malaria. In the north a much larger part, probably two-thirds of the reputed malarial mortality, represents unrecognized typhoid. Osler said long ago that north of the Mason-Dixon line every continued fever not yielding to quinin in six or seven days should be considered typhoid until definitely ascertained to be something else. I am led to believe that this mental attitude toward continued fevers is as becoming to medical men in the south as to those north of the Mason-Dixon line. Outside of certain well-known areas malaria does not kill citizens of the United States, and within those areas, where malaria is a cause of grave sickness, its right to be considered an important cause of death has not been subjected to the rigid scrutiny which its apparent importance demands. Speaking for that part of the United States which I know best, I can say that the terms "malarial" and "typhomalarial" express no definite belief as to the cause or character of a disease, but are applied to fevers of indeterminate nature, in the same way that some fourteen other titles found in the United States Census Reports are used—simply for want of a more satisfactory word.

To the large deficiency in the typhoid mortality which is fairly ascribable to the malaria delusion, one must add a further correction for the missed diagnosis of typhoid at the extremes of life. The accepted view that typhoid is unusual after middle life is not at present undergoing revision, so far as I know. But with respect to children the conviction is gradually settling in the minds of pediatrists that typhoid is common in childhood. At a recent meeting of the New York Academy of Medicine J. P. Crozer Griffith remarked on the steady change of medical opinion on this point. He said that in the Children's Hospital of Philadelphia the cases of typhoid constantly outnumber those of any other single disease, and that the superabundance of typhoid is often a cause of embarrassment in the instruction of students. New York pediatrists remarked that they seldom see

typhoid fever in children, but that the children of the metropolis often suffer with an obscure fever, which, for want of a better name, a distinguished clinician called "New York" fever. This circumstance reminds one of the fever that recurred year after year in a certain American city, and which, for want of a better name, was called "Ithaca fever."

When to these sources of error in the diagnosis of typhoid fever the ambulant and the anomalous cases are added, the total default in diagnosis is seen to amount to at least 50 per cent. of cases actually occurring. So long as we deceive ourselves thus grossly as to the amount of typhoid fever prevailing, we are not in a position to profit largely by our knowledge of its prevention. But there is another important point on which, if not deceived, we are only half informed; namely, as to the preferred habitat of typhoid fever.

The belief that typhoid fever is more common in close communities, and that its incidence is heaviest on cities, has its place in the medical mind by a sort of common consent. The grounds of the belief have not been examined, if indeed they have been stated. The late Colonel George Waring said in 1878: "Typhoid is peculiarly a disease of the country rather than of the town." No similar statement, I believe, is made by any American medical author; but the opposite view, that typhoid is peculiarly a disease of cities, is expressed in the most recent editions of at least four American text-books on medicine. Typhoid fever makes its most striking demonstrations in cities, and the definite influence of sanitary works on its prevalence has made the typhoid rate the accepted index of municipal hygiene. Typhoid fever has, nevertheless, at the present time and in this country, a heavier incidence on small communities and rural districts, and probably this has been the case for a long time; for sanitary works have neither such excellence nor so wide employment in American cities as to give urban populations in general

good defenses against typhoid. On the contrary, the conditions of American cities with respect to water supply and disposal of waste might be expected to produce a constantly higher typhoid mortality than is experienced under rural conditions.

The returns of the Twelfth Census show that the typhoid mortality per 100,000 of population in the registration area was slightly higher in the rural districts than in the cities (25.5 to 25.3). The "registration area" contains, however, but one-third of the total population of the country, and comprises the populations of 341 cities of 8,000 inhabitants and upward. But a small part of the aggregate population of the registration area is, therefore, classed as rural.

When we examine the rank of typhoid fever among all the causes of death we find that rural typhoid fever is credited with 62 in every 1,000 deaths, as against 38 per 1,000 for urban typhoid.

Taking the 21 grand groups into which the Census office divides the country on a basis of physiography, and arranging them according to population density, one finds that the typhoid mortality rises as population density falls until we reach the low density of 50 persons per square mile. Below this point the relations of typhoid are, as might be expected, quite irregular.

The Census divides the United States into five grand divisions, which, arranged in the order of their rural population, are as follows: The south central division having 88.9 per cent. of its total population rural, and a typhoid mortality of 79 per 100,000; south Atlantic division having a population 83 per cent. rural, and a typhoid mortality of 62 per 100,000; western division, population 68.8 per cent. rural, typhoid mortality of 33 per 100,000; north central division, population 59.4 per cent. rural, typhoid mortality of 42 per 100,000; north Atlantic division, population 41 per cent. rural, typhoid mortality of 30 per 100,000. Here we find the typhoid mortality rising as the urban population falls.

Some doubt may be admitted as to whether these grand divisions can fairly be compared. If the United

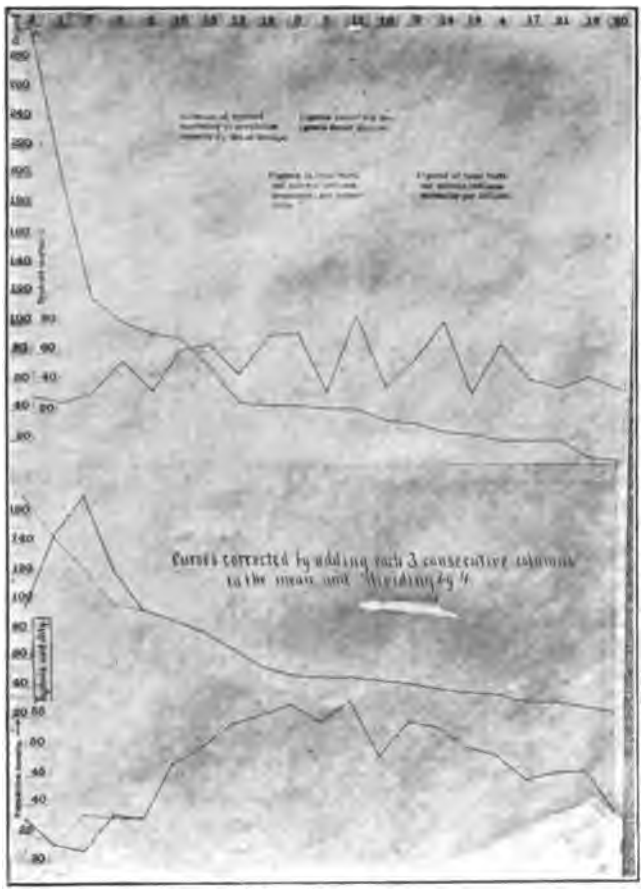


Chart 2.—Showing relation of typhoid mortality to population density. The figures across the tops indicate the Census Grand Groups in the order of their population density per square mile. The figures on the left vertical margin are the population densities. The figures in the inner left-hand column indicate the typhoid mortality.

States be divided into state groups on the basis of the distribution of population between town and country,

we can eliminate to a great extent the influence of latitude and longitude.

Thus there are five states whose rural populations are under 30 per cent. of the totals. They are Massachusetts, New York, Rhode Island, New Jersey and the District of Columbia. Their aggregate population is 12,665,183, of which number 70 per cent. are classed as urban, i. e., live in towns of 8,000 or over. Their combined typhoid mortality is 25 per 100,000. (See Table I.)

Six states have populations between 40 and 50 per cent. rural—Connecticut, Pennsylvania, Delaware, Maryland, Illinois and California. In these the total population is 50 per cent. urban, and the typhoid mortality for 1900 was 42 per 100,000.

Seven states have populations between 50 and 60 per cent. rural—New Hampshire, Ohio, Michigan, Wisconsin, Missouri, Colorado, Washington. Their population is 43 per cent. urban, and their typhoid mortality in 1900 was 38 per 100,000.

Eight states have populations between 60 and 70 per cent. urban—Maine, Indiana, Minnesota, Louisiana, Montana, Wyoming, Utah, Oregon. Their population is 25 per cent. urban and their typhoid mortality in 1900 was 46 per 100,000.

Twelve states have populations between 70 and 80 per cent. rural—Vermont, Virginia, South Carolina, Georgia, Florida, Tennessee, Alabama, Texas. Their population is 13 per cent. urban, and their typhoid mortality in 1900 was 62 per 100,000.

Twelve states have populations more than 80 per cent. rural—West Virginia, North Carolina, North Dakota, Mississippi, Indian Territory, Oklahoma, Arkansas, New Mexico, Arizona, Nevada, Idaho. Their population is 5 per cent. urban, and their typhoid mortality in 1900 was 67 per 100,000. Taking the twenty-one grand groups into which the country is divided on a basis of physiography, and arranging them according to their popula-

tion density, one finds that the typhoid mortality rises as population density decreases, until we reach a population density of 50 persons per square mile. Below that

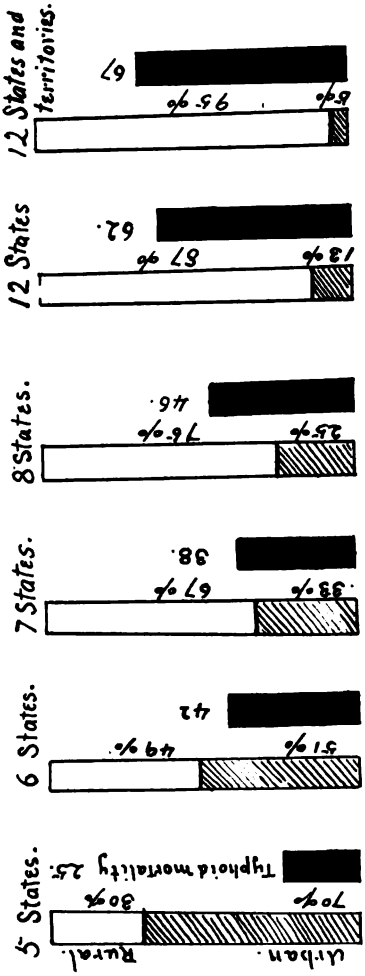


Chart 3. Showing typhoid increasing as we pass from urban to rural conditions. Rural populations indicated by white, urban by shaded areas. Black columns indicate typhoid mortality.

point the relations of typhoid to population density are, as might be expected, quite irregular. (See Table II.)

The experience of Maryland supports the general indications of these figures. There is but one large city in

the state. The inhabitants of Baltimore are half of the total population of the state. There are but two other towns having as many as 8,000 inhabitants. It may be fairly said that the population of the state is equally divided between rural and urban conditions. The ratio of typhoid mortality is 1 to 2.5 in favor of Baltimore, a poorly-sewered city, supplied with unfiltered water from an extensive and unprotected water-shed.

Dividing the cities of this country into ten classes on a descending population scale, we find that the typhoid mortality rises somewhat as populations diminish. Thus the least mortality in 1900 (24 per 100,000) fell on the three cities having populations of more than 1,000,000—New York, Chicago and Philadelphia. Not one of these cities is watered or sewered in a thoroughly modern way. Indeed, one of them is notoriously bad in respect to water supply.

There are but three cities in the half-million class—Boston, Baltimore and St. Louis—whose combined mortality for 1900 was 32 per 100,000.

Next we have five cities—Cleveland, Buffalo, San Francisco, Cincinnati and Pittsburg—whose average population is 344,891, and whose combined typhoid mortality for 1900 was 57. In this group we have the banner city of the year in respect to typhoid mortality among populations of over 100,000, Pittsburg, with the large rate of 147 per 100,000. The combined mortality in this class, if Pittsburg were omitted, would be 37 per 100,000.

Next are eight cities with an average population of 249,094, and a typhoid mortality for 1900 of 41 per 100,000. The city of Washington, in this class, had a mortality of 81 per 100,000, adding 6 points to the combined mortality of the class.

In the next class are eighteen cities whose average population is 128,281, and whose typhoid mortality in 1900 was 40 per 100,000. The second city of the coun-

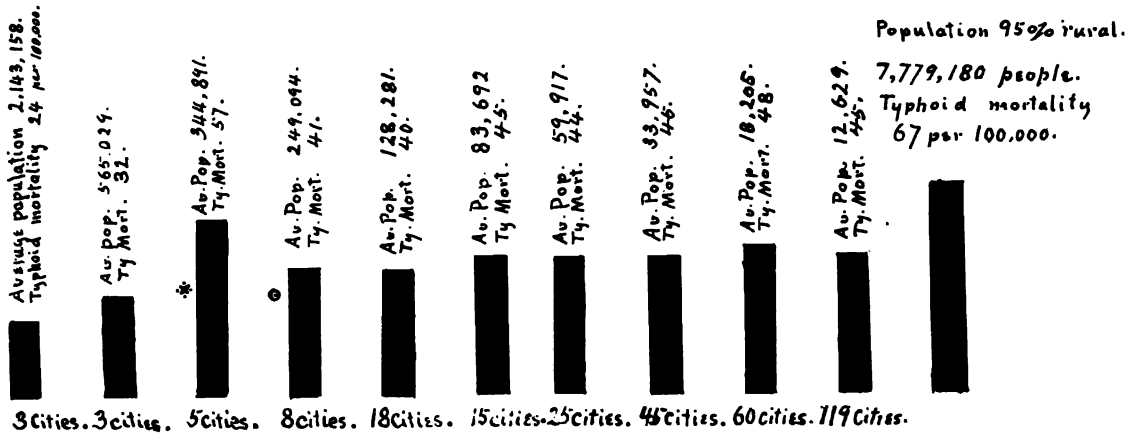


Chart 4. Typhoid in cities. Showing general inverse relation of typhoid to population.

* Includes Pittsburg with typhoid mortality 147 per 100,000.

⊙ Includes Washington with typhoid mortality 81 per 100,000.

try in typhoid mortality among populations of over 100,000 is in this class, Allegheny, Pa.

In the next class are fifteen cities with populations averaging 83,692, and typhoid mortality for 1900 was 45 per 100,000.

Twenty-five cities whose populations averaged 59,517 had a typhoid mortality in 1900 of 44 per 100,000. The heaviest individual rate of the year in populations over 50,000 fell in this class on Savannah; 107 deaths among her 54,000 citizens, a rate of 197 per 100,000.

In the next class are sixty cities having an average population of 18,205, whose combined typhoid mortality for 1900 was 48 per 100,000.

Finally there are 119 cities whose populations average 12,629, and whose combined typhoid mortality for the year was 45 per 100,000.

Of twenty-four towns having typhoid mortality rates above 100, the tenth class furnishes eleven in the states of Connecticut, Iowa, Indiana, Louisiana, Wisconsin, Michigan, Kansas and Illinois. (See Table IV.)

The ninth class furnishes five towns, in Kentucky one, in New York two, in Mississippi one, in Ohio one.

The eighth class furnishes four towns, in Pennsylvania two, in Ohio one and in Colorado one.

The seventh class furnishes two towns, one in South Carolina and one in Georgia.

The sixth class furnishes one city, Atlanta, Ga., eighteenth in order of typhoid rate descending.

The third class furnishes one city, Pittsburg, Pa., sixth in rank of typhoid mortality descending.

The states contributing the eight highest urban rates are, in order, Georgia, Louisiana, Connecticut, Kentucky, Iowa, Pennsylvania, New York. Pennsylvania has two cities in this list.

In the list of cities showing rates above 100, Ohio has three towns; Pennsylvania, two; Connecticut, two; Iowa, two; Illinois, two; Georgia, two; New York, two; Louisiana, one; South Carolina, one; Kentucky, one;

Indiana, one; Wisconsin, one; Kansas, one; Mississippi, one; Colorado, one; Michigan, one. (See Tables III and IV.)

The total population accounted for in this analysis of typhoid mortality is 20,922,886. The actual increase of typhoid mortality as the scale of population falls is probably greater than these tables indicate, for the states whose general mortality from typhoid is comparatively low are the chief contributors to the ninth and tenth classes, whose rates are highest.

The foregoing studies seem to warrant two conclusions, each having an important bearing on the general prophylaxis of typhoid fever. The first and more important is that the typhoid mortality reported in this country is not more (probably less) than 65 per cent. of the typhoid mortality actually occurring. Of the total morbidity from the disease a considerable part runs its course to recovery without coming under medical observation, and of the cases receiving professional care fully one-half are not recognized as typhoid fever. It therefore follows that the first step in the restriction of typhoid fever must fail for lack of diagnostic skill in the rank and file of the medical profession. The correction of this delinquency does not wait on improved methods of clinical or laboratory diagnosis, but on a general realization of the ubiquity and perennial activity of typhoid fever, and on the lumbering advance of reform in medical education.

A subsidiary conclusion from this part of the present study is that a very large majority of the deaths attributed to malaria are due to causes in which malaria does not even participate.

The second important conclusion, drawn from a study of the distribution of typhoid mortality, is that the infection is more heavily seeded in smaller communities, and that the propagation of the disease is in general from the country to the town, rather than from town to country. The explanation of this fact is probably found in

the greater care given to the disposal of human excrement as communities grow more populous.

The prophylaxis of typhoid fever consists essentially in defense against excremental contamination, and precisely as the barriers are more or less remote from the sources of infection is the defense less or more effective. In this view of the prevention of typhoid fever it must appear that, notwithstanding the very great value of the defenses on which cities most rely, these defenses do not, and by their widest conceivable extension can not, satisfy the requirements of a rational prophylaxis of typhoid fever. In town and country alike the appointed time and place for intervention against the spread of typhoid fever is whenever and wherever a case of typhoid fever is recognized. Those outbreaks of typhoid which from time to time scandalize great cities and bring down wrath on the authorities are, as a rule, less important than, and may be part of, unremarked, unopposed, or perhaps lie-coddled epidemics covering the countryside for miles around. To set up at the city gates expensive works for the purification of the public water supply as a main defense against water-borne disease is literally a preposterous arrangement. It waives the choice of time, place and weapons, conceding all these advantages to the enemy. It assumes that the vices of rural insanitation are invincible, but they are not. If there is one direction in which, more than another, the political influence of great cities should be exerted, it is to bring rural districts and small communities under legal obligation to render strict account of deaths and the causes of death, and to take and give good heed to the preventable diseases at the time and place of their occurrence.

TABLE I.

FIVE STATES WHOSE URBAN POPULATIONS ARE 60 PER CENT. AND UPWARDS.

	Population.	Urban.	Typhoid.
Massachusetts	2,805,346	2,132,623	625
Rhode Island	428,556	347,892	102
New York	7,268,894	4,980,042	1,776
New Jersey	1,883,669	1,153,001	398
District of Columbia	278,718	278,718	225
	12,665,183	8,892,276	3,126

Rural population, 30 per cent. Typhoid per 100,000, 25.

SIX STATES WHOSE URBAN POPULATIONS ARE BETWEEN 40 AND 60 PER CENT.

	Population.	Urban.	Typhoid.
Connecticut	908,420	483,068	249
Pennsylvania	6,302,115	2,865,937	2,779
Delaware	184,735	76,508	90
Maryland	1,188,044	557,497	510
Illinois	4,821,550	2,271,940	1,897
California	1,485,053	649,474	542
	14,889,917	7,593,424	6,067

Rural population, 49 per cent. Typhoid per 100,000, 42.

SEVEN STATES WHOSE POPULATIONS ARE FROM 30 TO 40 PER CENT. URBAN.

	Population.	Urban.	Typhoid.
New Hampshire	411,588	158,920	69
Ohio	4,157,545	1,599,840	1,795
Michigan	2,420,982	747,334	680
Wisconsin	2,069,042	634,437	365
Missouri	3,106,665	955,563	1,790
Colorado	539,700	205,703	222
Washington	518,103	165,282	171
	13,223,625	4,467,079	5,092

Rural population, 67 per cent. Typhoid per 100,000, 38.

EIGHT STATES WITH POPULATIONS 20 TO 30 PER CENT. URBAN.

	Population.	Urban.	Typhoid.
Maine	694,466	164,639	200
Indiana	2,516,462	607,834	1,496
Minnesota	1,751,394	470,046	386
Louisiana	1,381,625	314,386	1,077
Montana	243,329	65,623	42
Wyoming	92,531	22,294	16
Utah	276,749	69,844	73
Oregon	413,536	98,807	106
	73,700,092	1,813,473	3,396

Rural population, 75 per cent. Typhoid per 100,000, 46.

TWELVE STATES WITH POPULATIONS FROM 10 TO 20 PER CENT. URBAN.

	Population.	Urban.	Typhoid.
Vermont	343,641	305,054	107
Virginia	1,854,184	1,582,489	914
South Carolina	1,340,316	1,240,146	970
Georgia	2,216,331	1,972,562	1,585
Florida	528,542	449,413	290
Iowa	2,231,853	1,857,128	549
Nebraska	1,066,300	897,575	260
Kansas	1,470,495	1,265,288	688
Kentucky	2,147,174	1,784,115	1,669
Tennessee	2,020,216	1,750,298	1,709
Alabama	1,828,697	1,694,991	1,713
Texas	3,048,710	2,704,848	2,014
	20,096,459	17,503,907	12,468

Rural population, 87 per cent. Typhoid per 100,000, 62.

TWELVE STATES AND TERRITORIES HAVING POPULATIONS LESS THAN 10 PER CENT. URBAN.

	Population.	Urban.	Typhoid.
West Virgnia	958,800	885,197	586
North Carolina	1,893,810	1,797,273	1,250
North Dakota	319,146	309,548	65
South Dakota	401,750	391,484	73
Mississippi	1,581,270	1,510,176	1,370
Indian Territory	392,060	392,060	321
Oklahoma	398,331	378,288	201
Arkansas	1,311,564	1,240,201	1,172

	Population.	Urban.	Typhoid.
New Mexico	195,310	195,310	102
Arizona	122,931	122,931	41
Nevada	42,335	42,335	12
Idaho	161,772	161,772	37
	7,779,179	7,426,575	5,230

Rural population, 95 per cent. Typhoid per 100,000, 67.

TABLE II.

THE 21 GRAND GROUPS, ACCORDING TO THE TWELFTH CENSUS, SHOWING POPULATIONS, TYPHOID-MALARIA VARIETIES, WITH DENSITIES AND TYPHOID RATES.

Grand Group.	Typhoid Deaths.	Malaria Deaths.	Population.	Density.	Typhoid Mor'y.
1.	868	157	3,824,576	198.4	23
2.	1,900	694	7,139,889	299.8	27
3.	625	922	1,193,697	29.0	52
4.	1,111	1,179	1,767,487	16.5	63
5.	604	94	2,063,453	38.1	29
6.	991	108	3,249,040	89.0	30
7.	1,701	193	5,910,100	114.0	29
8.	3,796	557	7,488,008	99.4	51
9.	2,886	695	4,031,150	39.0	70
10.	1,774	309	3,018,359	85.7	58
11.	3,971	2,285	4,812,414	37.7	83
12.	753	1,424	1,090,623	39.0	69
13.	1,198	571	2,872,624	42.2	42
14.	4,319	3,597	5,424,490	21.9	79
15.	3,410	695	5,458,379	65.0	62
16.	2,645	641	8,133,937	30.6	33
17.	537	169	1,446,643	16.5	37
18.	580	199	1,442,684	3.4	40
19.	543	101	1,990,622	20.3	28
20.	638	220	1,965,065	2.2	33
21.	529	64	1,671,335	16.0	32

TABLE III.

Three hundred and one American cities classified on a descending scale of populations; showing typhoid mortality according to U. S. Census figures for each of ten classes.

CLASS I.

	Population.	Deaths from Typhoid.
Chicago	1,698,575	470
New York	3,437,202	594
Philadelphia	1,293,697	489
Totals	6,429,474	1,553

Average population, 2,143,158. Typhoid mortality per 100,000, 24.

CLASS II.

	Population.	Deaths from Typhoid.
Boston	560,892	153
Baltimore	508,957	177
St. Louis	575,238	190
Totals	1,695,087	520

Average population, 565,029. Typhoid mortality per 100,000, 32.

CLASS III.

	Population.	Deaths from Typhoid.
Cleveland	381,768	184
Buffalo	352,387	88
San Francisco	342,782	135
Cincinnati	325,902	106
Pittsburg *	321,616	474
Totals	1,724,455	987

Average population, 344,891. Typhoid mortality per 100,000, 57.

* Pittsburg being omitted, the typhoid mortality of the group is 37 per 100,000.

CLASS IV.—EIGHT CITIES.

	Population.	Deaths from Typhoid.
New Orleans	287,104	191
Detroit	285,704	54
Milwaukee	285,315	52
Washington, D. C.*	278,718	225
Newark, N. J	246,070	26
Jersey City	206,433	46
Louisville, Ky	204,731	150
Minneapolis	202,718	83
Totals	1,996,793	827

Average population, 249,094. Typhoid mortality per 100,000, 41.

* Washington being omitted, the typhoid mortality of the group is 35 per 100,000.

CLASS V.—EIGHTEEN CITIES.

	Population.	Deaths from Typhoid.
Providence	174,597	52
Indianapolis	169,164	77
Kansas City	163,752	125
Rochester	162,608	38
St. Paul	162,065	36
Denver	133,859	57
Toledo	131,822	53
Allegheny, Pa	129,896	134
Columbus, Ohio	125,560	68
Worcester, Mass	118,421	21
Syracuse, N. Y	108,374	25
Fall River	104,863	23
Los Angeles	102,320	51
New Haven	108,027	33
Paterson, N. J	105,171	31
St. Joseph, Mo	102,979	37
Scranton	102,026	30
Omaha	102,555	41
Totals	2,309,059	932

Average population, 128,281. Typhoid per 100,000, 40.

CLASS VI.—FIFTEEN CITIES.

	Population.	Deaths from Typhoid.
Albany	91,151	41
Richmond, Va	85,050	66
Dayton, Ohio	85,333	45
Seattle	80,671	31
Atlanta, Ga	89,872	98
Lowell, Mass	94,969	18
Cambridge, Mass	91,886	19
Portland, Ore	90,426	22
Grand Rapids, Mich	87,565	34
Hartford, Conn	79,850	45
Reading, Pa	78,961	35
Wilmington, Del	76,508	50
Camden, N. J	75,935	22
Trenton, N. J	73,307	25
Bridgeport, Conn	70,996	11
Totals	1,255,385	562

Average population, 83,692. Typhoid per 100,000, 45.

CLASS VII.—TWENTY-FIVE CITIES.

Total population	1,389,462
Average population	55,578
Total typhoid deaths	625
Typhoid mortality per 100,000	45

	Population.	Deaths from Typhoid.
Oakland, Cal	66,960	22
Lynn, Mass	68,513	13
Lawrence, Mass	62,559	8

	Population.	Deaths from Typhoid.
Springfield, Mass	62,059	18
New Bedford, Mass	62,442	16
Somerville, Mass	61,643	17
Elizabeth, N. J	52,130	5
Wilkesbarre, Pa	51,721	14
Erie, Pa	52,733	18
Harrisburg, Pa	50,167	20
Evansville, Ind	59,007	45
Charleston, S. C	55,807	70
Savannah, Ga	54,244	107
Manchester, N. H	56,987	6
Troy, N. Y	60,651	53
Utica, N. Y	56,383	3
Salt Lake, Utah	53,531	16
San Antonio, Texas	53,321	44
Duluth, Minn	52,969	23
Portland, Me	50,145	21
Hoboken, N. J	59,364	19
Yonkers, N. Y	47,931	6
Holyoke, Mass	45,712	13
Waterbury, Conn	45,859	21
Norfolk, Va	46,624	27
Totals	1,389,462	625

CLASS VIII.—FORTY-FIVE CITIES.

Total population	1,528,061
Average population	33,957
Total typhoid deaths	689
Typhoid mortality per 100,000	45

	Population.	Deaths from Typhoid.
Haverhill, Mass	37,175	4
McKeesport, Pa	34,227	15
Altoona, Pa	38,973	19
Spokane, Wash	36,848	19
Davenport, Iowa	35,254	23
Sioux City, Iowa	33,111	13
Johnstown, Pa	35,936	35
Elmira, N. Y	35,672	13
Chelsea, Mass	34,072	8
Springfield, Ill	34,159	19
Bayonne, N. J	32,722	5
Superior, Wis	31,091	19
Allentown, Pa	35,416	17
Salem, Mass	35,956	7
Youngstown, Ohio	44,885	51
Brockton, Mass	40,063	16
Binghamton, N. Y	39,647	13
Lancaster, Pa	41,459	23
Covington, Ky	42,938	17
Mobile, Ala	38,469	31
Wheeling, W. Va	38,878	26
Lincoln, Neb	40,169	14
Terre Haute, Ind	36,673	22
Saginaw, Mich	42,345	17
Tacoma, Wash	37,714	7
Fitchburg, Mass	31,531	6
Malden, Mass	33,664	13
Newton, Mass	33,587	7
Taunton, Mass	31,036	3
Auburn, N. Y	30,345	12
Schenectady, N. Y	31,683	10
Canton, Ohio	30,667	19
Pawtucket, R. I	39,231	7
Quincy, Mass	36,252	8
Sacramento, Cal	29,282	1
Woonsocket, R. I	28,204	6
New Castle, Pa	28,339	42
Jacksonville, Fla	28,429	19
Easton, Pa	25,238	4
Newport, Ky	28,301	15
Pueblo, Colo	28,157	31
Passaic, N. J	27,777	10

	Population.	Deaths from Typhoid.
Williamsport, Pa	28,757	11
Bay City, Mich	27,628	8
Gloucester, Mass	26,121	4
Totals	1,528,061	689

CLASS IX.—SIXTY CITIES.

Total population	1,092,281
Average population	18,205
Total typhoid	521
Typhoid per 100,000	48

	Population.	Deaths from Typhoid.
Norristown, Pa	22,265	9
Jamestown, N. Y	22,892	16
Muncie, Ind	20,942	10
Burlington, Iowa	23,201	14
Wilmington, N. C	20,976	10
Leavenworth, Kan	20,735	8
Wichita, Kan	24,671	17
New Brunswick, N. J	20,006	4
Jackson, Mich	25,180	16
Kalamazoo, Mich	24,404	7
Muskegon, Mich	20,818	4
Bangor, Me	21,850	13
Petersburg, Va	21,810	18
Cohoes, N. Y	23,910	35
Watertown, N. Y	21,696	18
Lafayette, Ind	18,116	6
Richmond, Ind	18,226	10
Ottumwa, Iowa	18,197	18
Northampton, Mass	18,643	6
Chicopee, Mass	19,167	9
Medford, Mass	18,244	7
Lynchburg, Va	18,891	17
Key West, Fla	17,114	7
Paducah, Ky	19,446	31
Lebanon, Pa	17,628	8
Eau Claire, Wis	17,517	5
Battle Creek, Mich	18,563	7
Port Huron, Mich	19,158	7
Concord, N. H	19,632	2
Niagara, N. Y	19,457	24
Findlay, Ohio	17,613	19
Portsmouth, Ohio	17,870	9
Sault Ste. Marie, Mich	19,158	1
Natchez, Miss	23,898	27
Orange, N. J	24,141	5
Perth Amboy, N. J	17,699	2
Kingston, N. Y	24,535	5
Newburgh, N. Y	24,943	10
Amsterdam, N. Y	20,929	3
Gloversville, N. Y	18,319	3
Poughkeepsie, N. Y	24,029	7
Hamilton, Ohio	23,914	4
Lima, Ohio	21,723	8
Newark, Ohio	18,157	5
Central Falls, R. I	18,167	2
Newport, R. I	22,034	12
Burlington, Vt	18,640	4
Santiago, Cal	17,700	2
Meriden, Conn	24,296	5
New London, Conn	17,548	3
Norwich, Conn	17,251	1
Belleville, Ill	17,484	14
Aurora, Ill	24,147	10
Decatur, Ill	20,754	8
Galesburg, Ill	18,607	4
Everett, Mass	24,336	6
Winona, Minn	19,714	5
Madison, Wis	19,164	3
Green Bay, Wis	18,684	6
Nashua, N. H	28,898	1
Totals	1,092,281	521

CLASS X.—ONE HUNDRED AND NINETEEN CITIES.

Total population	1,502,829
Average population	12,629
Total typhoid	674
Typhoid per 100,000	45

	Population.	Deaths from Typhoid.
Lockport, N. Y.	16,581	3
Shreveport, La.	16,013	30
Marinette, Wis.	16,195	19
Appleton, Wis.	15,085	3
Lansing, Mich.	16,485	4
Biddeford, Me.	16,145	13
Alameda, Cal.	16,464	5
Rome, N. Y.	15,343	6
Helena, Mont.	10,770	4
Meadville, Pa.	10,291	10
Carbondale, Pa.	13,536	5
Columbia, Pa.	12,316	6
Pottstown, Pa.	13,696	5
South Bethlehem, Pa.	13,241	6
Oil City, Pa.	13,264	11
Rutland, Vt.	11,499	4
Harrisontown, N. J.	10,596	3
Millville, N. J.	10,583	5
Montclair, N. J.	13,962	1
Phillipsburg, N. J.	10,052	2
Ithaca, N. Y.	13,000	4
Flint, Mich.	13,103	5
Ann Arbor, Mich.	14,509	3
Marquette, Mich.	10,058	8
Menominee, Mich.	12,818	15
West Bay City, Mich.	13,119	6
Dover, N. H.	13,207	3
Portsmouth, N. H.	10,637	1
Augusta, Me.	11,683	5
Fresno, Cal.	12,470	5
Bridgeton, N. J.	13,913	3
Morristown, N. J.	11,267	3
Watervliet, N. Y.	14,321	12
Dunkirk, N. Y.	11,616	6
Geneva, N. Y.	10,433	5
Lansingburg, N. Y.	12,595	8
Ogdensburg, N. Y.	12,633	11
Saratoga, N. Y.	12,409	6
Ashtabula, Ohio	12,949	5
Chillicothe, Ohio	12,976	9
Tiffin, Ohio	10,989	4
Massillon, Ohio	11,444	5
Marietta, Ohio	13,000	8
Lawrence, Kan.	10,862	3
Phœnixville, Pa.	9,196	4
Bennington, Vt.	5,656	2
Barre, Vt.	8,448	3
Owosso, Mich.	8,696	4
Pontiac, Mich.	9,769	1
Escanaba, Mich.	9,549	9
Iron Mountain, Mich.	9,242	1
Ironwood, Mich.	9,705	4
Traverse City, Mich.	9,407	3
Berlin, N. H.	8,886	3
Laconia, N. H.	8,042	2
Rochester, N. H.	8,466	0
Keene, N. H.	9,165	0
Rockland, Me.	8,150	1
Hutchinson, Kan.	9,379	11
Carlisle, Pa.	9,626	5
Plymouth, Pa.	13,649	7
Glen Falls, N. Y.	12,613	5
Jeffersonville, Ind.	10,774	10
Columbus, Ind.	8,130	3
Peru, Ind.	8,463	10
Vincennes, Ind.	10,249	8
Keokuk, Iowa	14,641	15

	Population.	Deaths from Typhoid.
Muscatine, Iowa	14,073	13
Marshalltown, Iowa	11,544	18
Oskaloosa, Iowa	9,212	4
Leadville, Colo	12,455	6
Raleigh, N. C	13,643	6
Newburyport, Mass	14,478	4
Weymouth, Mass	11,324	1
Marlboro, Mass	13,609	1
Alexandria, Va	14,528	11
Annapolis, Md	8,525	2
Frederick, Md	9,296	4
Hudson, N. Y	9,528	5
Cortland, N. Y	9,014	5
Bellaire, Ohio	9,912	7
Middletown, Ohio	9,215	1
Warren, Ohio	8,529	6
Plainfield, N. J	15,369	2
Middletown, N. Y	14,522	3
Port Jervis, N. Y	9,385	3
Peekskill, N. Y	10,358	0
Corning, N. Y	11,061	5
Johnstown, N. Y	10,130	3
Olean, N. Y	9,462	3
Ironton, Ohio	11,868	12
DuBois, Pa	9,375	4
Hazeltown, Pa	14,230	3
Mahanoy, Pa	13,504	3
Mt. Carmel, Pa	13,179	2
Pittston, Pa	12,566	2
Pottsville, Pa	15,710	3
Steelton, Pa	12,086	3
Ansonia, Conn	2,681	2
Danbury, Conn	16,537	3
Greenwich, Conn	2,420	3
Middletown, Conn	9,589	5
Naugautuck, Conn	10,541	5
Norwalk, Conn	6,125	2
Stamford, Conn	15,997	1
Stonington, Conn	2,278	1
Wollingford, Conn	6,737	3
Bristol, Conn	6,268	11
Tarrington, Conn	8,360	2
Danville, Ill	16,354	17
Jacksonville, Ill	15,078	10
Ottawa, Ill	10,588	3
Michigan City, Ind	14,850	3
Beverly, Mass	13,884	4
Melrose, Mass	12,962	6
Mankato, Minn	10,599	6
Chippewa Falls, Wis	8,094	3
Beloit, Wis	10,436	0
Manitowoc, Wis	11,786	3
Totals	1,502,829	674

TABLE IV.

THE 24 CITIES HAVING TYPHOID MORTALITY RATES OF ABOVE 100 PER 100,000 IN THE DESCENDING ORDER.

	Population Class.	Typhoid Mortality per 100,000.
Savannah	7	197
Shreveport	10	187
Bristol, Conn	10	175
Paducah, Ky	9	159
Marshalltown, Iowa	10	156
Newcastle, Pa	8	147
Pittsburg, Pa	3	147
Cohoes, N. Y	9	146
Charleston, S. C	7	125
Greenwich, Conn	10	124
Niagara, N. Y	9	123

	Population Class	Typhoid Mortality per 100,000
Peru, Ind.	10	118
Marinette, Wis.	10	117
Menominee, Mich.	10	117
Hutchinson, Kan.	10	117
Youngstown, Ohio	8	114
Natchez, Miss.	9	113
Pueblo, Colo.	8	110
Atlanta, Ga.	6	109
Findlay, Ohio	9	108
Allegheny, Pa.	7	106
Danville, Ill.	10	104
Keokuk, Iowa	10	102
Ironton, Ohio	10	101

DISCUSSION.

DR. J. H. MUSSER, Philadelphia—It is fortunate that we can make an early, or more precise, diagnosis by modern bacteriologic methods, such as the employment of the serum test, or by culture methods; by such means we can make such precise diagnoses that there can hardly be any mistakes made regarding the nature of the prevailing fever or epidemic. There is no excuse, therefore, except probably in some communities or districts, for not making an early diagnosis. Unfortunately, however, there is a lack, apparently, of conscience in physicians. They are not awake to the dangers of the infection in cases of typhoid fever. Not long ago, within the last three years, I saw three cases of typhoid fever in one family in the country that had their origin as follows: The original case was an imported one. This man became infected in a near town and came to the country home of the physician, who was very careless regarding the disposal of the excreta. The relation of the privy was such that its contents drained into a driven well. This, of course, was the source of infection in the members of the family. One member of this family died and two were placed in great peril. These people were taken ill and all were sick, including the physician, due to the carelessness, or lack of conscience, on the part of the physician regarding the disposal of the excreta. There was a lack of regard for the protection of the people in that district. I could tell of many local epidemics arising through the faults of physicians. It is, therefore, necessary to take a lesson to ourselves before we preach to communities. Communities should be educated in regard to this. Above all, the civil authorities should be brought to realize their responsibilities. In cities where there are piles of dirt, etc., a policeman goes and places a red lantern at each pile to indicate danger; yet no civic authorities would place a red light danger signal in places where there were epidemics or possibilities of typhoid fever. Why is this done in the first instance? Because the city knows that if an accident should happen the authorities would be held responsible. If we should hold the civic authorities similarly responsible in cases of typhoid fever we

could soon get control of the disease. How can we help ourselves regarding this? It is a difficult problem and a difficult question to answer.

It has been said that the busy practitioner has not time to act as police and see that his people carry out his directions regarding the disposal of excreta, etc. We should see developed a conscientious sense of duty which should inspire the untiring effort of all to protect communities as well as individuals. Each practitioner in a community should have a trained assistant. Let him take a man, for instance, from the Johns Hopkins Hospital Laboratory to assist him in laboratory work; any good practitioner can afford this. I am satisfied that any practitioner of medicine who takes an assistant to do work of this character, and so shows the community that he is practicing the highest type of medicine and making every effort to protect the community, will increase his income to twice what it would have been without an assistant in charge. If, however, that does not seem to be practical, then a group of physicians should have an assistant to make diagnoses and watch the excreta and to determine whether a typhoid fever patient should be sent forth in a community with safety by examining the urine, etc. If this is not practical, then we should resort to government control. We must educate the civic government itself regarding the danger of typhoid fever patients, and make them understand that they should be under their charge. The physician should continue to treat the patient, but the civic authorities should be held responsible for him until he is perfectly harmless. The patient should not be allowed away from a community. All the excreta and discharges should be taken care of. Then, too, perhaps, the government should be held responsible for the laboratory diagnosis and also for the sanitary management of the case, absolutely from the start of the infection. I do not think this is an unreasonable thing to expect. When we realize the trouble the government is taking with some infections, when we realize the mass of money that is spent to prevent the spread of yellow fever or the plague, when we think of how many more lives are lost through the ravages of typhoid fever, and when we think that our hands are practically paralyzed in our attempts to obtain governmental control of these cases, we must marvel at it. They should look after typhoid epidemics as they do after other epidemics.

I had hoped to learn a ready method for disinfecting the urine. Disinfection of the urine is very essential before the patients get from out of our control. Probably we can make the urine sterile by the use of urotropin. In the later stages of the disease and throughout the period of convalescence and until a trained assistant or a government expert shows the urine is free from bacilli these patients should not be lost

sight of. The use of urotropin is one of the best methods for preventing as well as curing a complication of typhoid fever, i. e., cystitis; sterilizing the urine is the best aid in the way of prophylaxis.

Dr. DeLancey Rochester, Buffalo—Milk may be infected after leaving the farm, and the source of infection be difficult to trace. In Buffalo, in one of the healthiest districts, ten years ago, four members of the same family were taken ill with typhoid fever within four days of each other, and there was apparently no local reason for it. In looking for the source of the disease the milk supply was investigated. At the farm from which the milk came there was no typhoid. The milk was followed to the city, and the source of infection was found to be at the home of the man who delivered the milk. In his home was a case of typhoid fever; after he received the milk from the farm he had it bottled at his home, the same persons washing the bottles and caring for the typhoid patient. Thus it was accounted for. In his route through the city were found cases of typhoid fever.

In reference to the prevention of typhoid fever, we must remember that we do not know how long organisms persist in the stools after the patient has recovered from the attack. This is one chief source for the spread of the disease which should be borne in mind; after the patient recovers and goes about his business he still may be a source of infection on account of the persistence of the organisms in the stools.

I did not understand whether Dr. Fulton said that the disease was more prevalent in the country than in the cities, or whether the mortality was greater. If the latter, I think it is due to the lack of facilities, for the farmer will not or can not get the necessary nursing for these patients, and the consequence is that they die because they are not properly cared for; in such cases, of course, the physician is not at all at fault. I think the source of mortality in the country districts is to be found in this lack of proper care, or a lack of ability or willingness to care for these patients.

With regard to the disinfection of the urine, Dr. Musser has already referred to the use of urotropin. In three cases of typhoid fever we found the urine free from organisms and apparently in a sterile condition two weeks after the patient had recovered from the disease after giving urotropin; before that time the urine was infected.

With regard to disinfection of the stools, I have found that powdered chlorid of lime thoroughly mixed with each stool is efficacious.

Dr. Wm. Krauss, Memphis, Tenn.—We have a municipal ordinance which requires that every case of typhoid fever be reported, and in accordance with that requirement I reported a case. A lay inspector called and asked if the party had used

well water or cistern water and got the address of the milk man. He left no literature or instructions as to the hygienic management of the case. The patient, a young lady, was very fond of ice cream, and would send to a nearby factory for a little paperboxful perhaps once or twice a day, and this was probably the source of infection. I think a municipal health board culpable for entrusting such matters to a policeman. The inspector should be a specially trained medical man.

About ten years ago I kept the records of the board of health in Memphis, and in one year I noticed that the returns for the year were about 270 deaths from malarial fever and 35 deaths from typhoid fever. This was before our public water supply was from artesian wells. Surely this was a wrong proportion of deaths from these two fevers, for in one season I had in my service at St. Joseph's Hospital about 400 cases of fever, in the neighborhood of 260 of which were malarial, of which latter only one died. Most of these patients were from the swamps about Memphis and were in a very bad condition. This would argue that many of the 270 deaths on the records must have been typhoid fever, since malarial fever with us is not so fatal.

Dr. Frank Warner, Columbus, Ohio—It is doubtless true that some cases of typhoid fever develop as a result of an infection acquired in some other way than drinking water; by eating some raw vegetables which have on them some typhoid bacilli, or by any of the several but unusual methods of acquiring the disease. It remains a fact, however, that the number of cases which develop in any other way than by drinking infected water are very few indeed. Then, as this is the usual method of infection, it becomes the duty of physicians everywhere to urge better water supplies in their respective communities. Not only does it become our duty to speak of these things, but also to point out from time to time that the great underlying cause of typhoid fever is the result of an absolute indifference in various communities of the dangers arising from drinking the water that is ordinarily furnished by many municipalities. There needs to be a rude awakening from the absolute indifference to which many communities have fallen. If the people in a community really make up their minds that there is a real danger in an unfiltered public water supply the politicians who operate the places will quickly install the most modern and approved methods of purifying water. They will give any community just as good water as they demand, and no better. It seems to me that it is time we should seek to prevent this disease on a greater scale than mere study and prevention of individual cases. This prevention on a large scale can best be accomplished by the construction of public filtered water supplies. And this filtered water is rendered purest by sand filtration. It is frequently argued against

sand filtration that it does not remove all the bacteria of a disease-producing character. This is entirely true. It does remove some 98 or 99 per cent. of the pathogenic germs. Dr. William H. Welch has stated that one is not so likely to develop typhoid fever from drinking water which is only slightly infected with the typhoid germs as where it contains a large number of them. Then, if sand filtration does not remove all germs, and consequently all danger of infection, as it does remove at least 98 per cent. of the germs, it removes an equal per cent. of the danger of infecting the individual drinking the water. The ideal filter plant would remove all germs of a disease-producing character. Inasmuch as this can not be done, we must be content for the present to attain such a great improvement in the purity of a water in a community. And just in proportion that a pure water is furnished to a community, in just that proportion are we meeting the chief indication of prevention of typhoid fever.

Again, I believe that securing of a better water supply in any community should be a part of the labor of the physician and bacteriologist as well as the engineer. And this labor of the physician and bacteriologist should be as liberally paid for as the labor of the engineer. In seeking a water supply the engineer should be aided by the bacteriologist. This aid should not consist, as it does so many times when the engineeer calls such help, in simply examining the water for a time or two, but careful bacteriologic examinations should be made over a considerable period; these examinations should be made under varying conditions, so that a reliable conclusion may be drawn. While it is true that some of the more able sanitary engineers cover these points, still it is the rule that in the establishing of a public water supply the engineer regards it as distinctively an engineering problem rather than a joint undertaking of the engineer and the bacteriologist and physician. When this fact becomes better known and recognized we shall have purer water supplies and less typhoid fever.

Dr. J. N. Hurty, Indianapolis—It seems to me that the question of prophylaxis goes back further than we teach. At first we thought that, after the etiology of the disease had been discovered, all we had to do was to disinfect the dejecta. Now we find that there are clinicians and diagnosticians who do not make out all cases of typhoid fever. We all know that the dejecta carry the typhoid organisms long after the patients are seemingly well, months after in some cases. This fact should teach us something. We must not only disinfect the dejecta, but we must properly dispose of it; if that be done then we will soon abolish typhoid fever. I believe that all intestinal disorders, the germs of which are cultured in the intestines, will spread under certain conditions

in the towns and large cities unless we treat and do away with the dejecta. We must filter the water in our towns and cities; and we must see that the dejecta are cared for in a sanitary way, then typhoid fever will certainly be abolished. I have read there was practically no sanitation in the days of Henry VIII, and that at that time the dejecta and slops were thrown into the streets; the proposition to care for them in pits was received with scorn and ridicule. But when the streets were used no longer for dejecta disposal there was a pronounced diminution in the number of cases of disease that arose from such causes; the death rate of typhoid fever and other diseases fell, and health conditions improved, after the human dejecta were cared for in pits. Let us go deeper into the study of this question and let us learn to care properly for all dejecta; then I can promise that the disease will disappear.

Dr. Henry Sewall, Denver—In the prophylaxis of tuberculosis it is admitted that physiologic resistance of the individual to the growth of tubercle bacilli is only second in importance to the exclusion of the bacilli themselves. In the prophylaxis of typhoid fever all sanitary effort seems to be restricted to the exclusion of the germ, and we hear nothing of the means of increasing the vital resistance of the individual after the manner which, in the conflict with tuberculosis, has proved so effective. The development of typhoid fever in a person is very largely determined by his physiologic condition while the typhoid germs lie in his intestines. A man recently under my charge had a mild run of fever for a few days; the Widal test was negative and the attack seemed clearly of "functional" nature. He was allowed to get up and increase his diet after some ten days, when shortly he became seriously ill, the blood test became positive and he nearly died with typhoid fever. Again, a little boy declared himself to have been perfectly well until he ran a foot race, when he was suddenly seized with vomiting and abdominal pain, ushering in a well-marked attack of typhoid fever. In each of these cases the attack of typhoid was exacerbated or precipitated as the result of physical exertion. A large proportion of our typhoid patients come from the hardiest and healthiest class of people. But they fall victims to the disease while engaged in work which upsets their physiologic equilibrium. They are very apt to be compelled to work to fatigue in the intense heat of the sun. It is not hard to imagine that under such conditions the germicidal powers of the gastric juice might deteriorate and the vital resistance of the body fluids and tissues themselves might be reduced. We might, therefore, speak of every individual enjoying, as regards typhoid fever, a certain immunity on a sliding scale. With every physiologic function doing well its co-ordinated task, the typhoid germ may possibly be ingested with impunity; immunity is high. But let

the vital machinery falter ever so little and infection readily occurs; immunity is low.

Dr. Deering J. Roberts, Nashville, Tenn.—Medical science is based on fact and on proof. Theories in medicine arise, spring up, live or die, depending on that idea. You can not build theories and make facts to fit them. Dr. Happel gave you the results of his observations; our work here is the result of observations. He records his observations of facts in his locality. On the other hand, representatives of Johns Hopkins and the University of Pennsylvania tell what they do not see. The microscope will tell you a fact and give the clinician aid when sustained by clinical observations. These men come with statements; Dr. Happel comes with observations. Take the humblest court in the land and submit to the bench two or three credible witnesses and you will be able to establish an observation as a fact. There is one witness from Arkansas. Dr. Witherspoon of Tennessee tells you what he saw. I claim to have seen the same fever for forty years. The Surgeon-General of the United States saw in the Army reports during the sixties something that was not down in the text-books, and he essayed to find out about it, only he went a little bit further and gave it a name. His efforts were sterile, so much so that he took back what he said regarding this fever and said he did not say it. Typhoid-malarial fever belongs to the ages of the past. One gentleman came here and endeavored to prove by therapeutic demonstration that we had a typho-malarial fever, "this and nothing more." You must look at things as they are and not as you wish to have them. I have seen 40 consecutive cases of fever in one season. Five years ago, before the Tennessee State Medical Society, I gave the clinical notes of a fever, "this and nothing more." This was not enteric fever. It ran along and gave an erratic temperature. Some have called it paratyphoid and atypical typhoid, etc. A leopard may change its spots, but still it is a leopard. There are certain characteristics about it which you can always recognize. What constitutes typhoid fever? The bacillus of Eberth must be present. Let the gentlemen with their monoculars and binoculars say what they please; we have the same in the laboratories in Tennessee. We have a series of recorded cases reported and published in the state medical society's transactions, with carefully trained observers, teachers in laboratory work, and in this series of cases the bacillus of Eberth was wanting. We have had it stated by able clinicians that this is a fever with erratic temperatures; that it was not attended by the abdominal symptoms of typhoid fever. I have seen 40 cases lasting 14 days or less, all of the same type. There have been reported 90 successive cases and none went beyond two weeks' duration. This is nothing but fever; let it alone and the patient will get well.

DR. C. F. ADAMS, Trenton, N. J.—We have flies and mosquitoes in New Jersey; the mosquitoes are growing less, so is malaria. The Delaware river is a large sewer, and those who live along its banks become infected with typhoid fever. We make our differential diagnosis by taking a drop of blood on a slide for the Widal reaction, and also smear a cover slip for the plasmodium, and mail them to the state laboratory, where they are examined free. We should like to have the government prohibit the use of unfiltered Delaware River water for drinking purposes.

DR. JAMES TYSON, Philadelphia—I have been surprised that no reference has been made to the easiest and simplest way of making drinking water innocuous, i. e., by boiling. In Philadelphia to-day our hospitals are filled with typhoid fever cases largely to the exclusion of others, and I am sure that very few of these need have had typhoid fever if the drinking water had been boiled. Of course, we must look back to the source of infection. There are times when no filtration will protect the water against the typhoid bacillus; in fact, I believe that it is considered impossible to eliminate entirely the bacillus even when we have the best filtration plants; especially in times of great freshets, when there is a blocking up of the apparatus. If the working people could be taught to use no drinking water that had not been boiled many epidemics would be averted. Of course, the difficulty of doing tnis is evident, but we as physicians could do much by insisting on this practice, not only among the poor, but among the rich as well, and with all with whom we come in contact. The Schuylkill river water has not a good reputation, but I know of no instance where the disease has been acquired through the use of this drinking water after it was boiled. Boiling will effectually destroy the typhoid bacillus.

DR. JAMES J. WALSH, New York—A prominent sanitary officer once said that when somebody had typhoid fever somebody ought to be hung. We know that we can contract typhoid fever only from a typhoid fever patient. If that typhoid fever patient is not properly cared for somebody is to be blamed for criminal negligence. In large cities what is to be done? Take a certain large city, not far from New York, which has had typhoid fever constantly in its midst for the past ten years. Every year there are a large number of deaths and the "boss" of that city has made the boast that he kept back the sanitary plant for a long time because provision had not been made for giving a part of the work to a friend of his. In this city there have been 3,000 deaths during that time from typhoid fever and nothing has been done toward getting a better and purer water supply. This boss has no sense of civic duty. Sanitary plants have been used in Germany for years; in Berlin proper filtration plants had been used thirty years

before they had a death from typhoid fever. People on the farms have less death rate than in the city.

With regard to filtration, the little town of Altoona, near Hamburg, did not have a single death from cholera in spite of the fact that they used the water from the Elba. When this disease is communicated to another some one is criminally responsible. We should make the municipal authorities understand that the dangers lie in allowing certain tyrants to continue in control in sanitary work when there is a prolonged outbreak of typhoid fever.

DR. WALTER SHROPSHIRE, Yoakum, Texas—I am surprised at Dr. Tyson's statement that his patients might have avoided typhoid fever by boiling their drinking water, suggesting water as the only means of conveying the disease. In the town where I live, with a population of 6,000, there are 50 or more cases of typhoid fever each year. These people without exception use water from an artesian well, 1,400 feet deep, which is pumped direct into the mains and is never exposed to the atmosphere till drawn from the hydrant for drinking. If these people get typhoid fever through their drinking water I am unable to see how the bacilli get into it. Two years ago there was a case of typhoid fever near me treated by one who did not have the dejecta properly cared for, permitting it to be thrown out on the ground surface, this being in early summer when the ground was dry and dusty. Immediately following this case I treated 15 others within a radius of 300 yards of it, whose infection was traceable to this focus, and they could not have obtained the poison through water, for it was drawn from the hydrant and used immediately. I recall an experience of 12 years ago, when I treated a case of typhoid in December, and was unable to have any disinfectant used at all. The nurse simply threw the dejecta out in the back yard. During the balance of December, January, February, March and April we had no typhoid fever in that neighborhood. During May we had a drouth and the surface became dry and dust was prevalent, being carried by a prevailing southeast wind. The dry weather had existed not more than two weeks when typhoid began developing around this focus, and I treated 73 cases within a radius of 300 yards of it; and seven-tenths of them were to the northwest where the prevailing wind carried the dust from this infected focus. These people all used different water from surface wells about 70 feet deep and through a heavy clay and light rock strata. Now, if these people were infected through their drinking water why were there no cases through the five rainy months, when the earth surface was wet and why such preponderance of cases to the northwest where the prevailing wind would carry the dust from this infected focus? And if it be conceded that the

spores were carried in dust by the wind and infected their drinking water, why could it not as well infect articles of food and thus reach the intestines of the individual? I am convinced that the dust was the means of infection in these cases as well as in many others. Again, our Spanish-American war demonstrated conclusively that flies can and do carry the infection, and Dr. Alice Hamilton of Chicago read a paper before this Association a year ago forcibly portraying the fact that flies do carry the infection. With such facts before us I think even Dr. Tyson should modify his remarks on the subject.

DR. T. J. HAPPEL, Trenton, Tenn.—The discussion of this morning has been on typhoid fever, not on the paper read by me on Tuesday.* The object of my paper was not to discuss the subject of typhoid fever, but to report cases of a fever occurring in my practice, which were cases of neither a typhoid nor a malarial fever.

I do not claim that there is no typhoid fever in the section of Tennessee in which I live, but in my own town it has almost entirely disappeared. We have not had a case of it for years. We account for our peculiar exemption from the fact that all of our drinking water is filtered through a vast bed of sand underlying our town. Some geologist claims that we are located over what in ages past was a large body of water which has been replaced by sand, and through this sand of an unknown depth our water filters, rendering it almost perfectly pure. While in our immediate neighborhood we have no typhoid fever, yet in certain sections of our county typical cases of typhoid fever are found.

When the cases reported to me are referred to as "atypical," "paratyphoid," etc., you are begging the question. In reference to microscopic examinations in these cases, I stated that examinations had been made in 138 cases of this fever by persons who had the reputation of being skilled in the use of the microscope and in laboratory work. Thus my clinical observations were not left unsupported. One person discussing the paper* suggested that the variations in temperature were due to the use of the coal-tar derivatives. The coal-tar products are never used by me except in those cases where I have failed to reduce the temperature by water and other febrifuges. In not one of the 100 cases which have come under my care has there been any tendency to hemorrhage. One member of the section referred to the fact that he had cured cases of this type by a certain routine medication, but my paper referred only incidentally to the subject of treatment. In the closing lines of the paper I mentioned the fact that I used guaiacol

* See page 31 of this volume.

carbonate in treating these cases the last season. If cases of this type of fever are let alone and nursed well they recover without or in spite of medicine. One of my patients recovered who was given by her mother on one occasion a box of sardines and allowed to eat them all at one time. At another time this same child during the third week of her fever was fed enough biscuits to have surfeited a well person. Had this been a case of typhoid fever we would probably have had a funeral. I have treated two cases of this fever in pregnant females, one five months advanced, the other seven. Both miscarried, and on the day following the miscarriage the temperature in both cases fell to normal and never went above that point again. Both made uneventful recoveries, a very different result so far as my experience has gone with typhoid fever under similar circumstances.

I wish again to emphasize the fact that the cases reported by me did not present a single characteristic symptom of typhoid fever.

Dr. John S. Fulton, Baltimore—I regret that any one should have misinterpreted my demonstration of the higher mortality of typhoid in rural districts as a reflection on the rural physician's skill. I know nothing about the comparative case mortality as between town and country, and I assume that the higher absolute mortality in the rural districts means a correspondingly higher morbidity. No other conclusion can be drawn from the evidence. In treating cases of typhoid fever the city physician has, so far as I know, but a single advantage over his fellow in the country, and that advantage is in skilled nursing. The trained nurse may, perhaps, profit the case mortality by the 1 or 1.5 per cent., but in my opinion the chief profit of trained nursing lies in the control of morbidity.

I take occasion to say now what I did not say in my paper, that the trained nurse, proportionately to the numerical strength of her profession, opposes more effective barriers to the spread of typhoid than does the practicing physician. That may be construed as a criticism of the profession and may be added to the other criticism of a 48 per cent. default of professional skill in the diagnosis of typhoid.

I did not intend in my paper to touch the minutiæ of prophylaxis, but I am glad that the discussion brought out the very central problem of disinfecting stools. A disinfectant for fecal matter should be first effective, and after that must be cheap, rapid, safe and convenient. No agent yet proposed meets all these requirements or even a majority of them. I am inclined to regard quicklime or air-slaked lime as the most generally available disinfecting agent for typhoid excreta.

But above all things I want to impress the Association with

the testimony of the profession itself, shown here in graphic form, and supporting, as I think, in the most convincing manner and for the whole United States, the testimony of the U. S. Army Commission that the medical men of the land habitually misinterpret the continued fevers of the United States. This is a definite and insistent challenge at the very threshold of the prophylaxis of these fevers. What will you do about it?

SOURCES OF PLAGUE.

W. J. CALVERT, M.D.
Department of Clinical Medicine, Missouri State University.
COLUMBIA, MO.

This subject involves a discussion of the early and present pandemics, cardinal features of the viability and dissemination of *Bacillus pestis* and the transmission of plague.

HISTORICAL SKETCH.

While indefinite statements regarding plague are found in the earliest writings, no definite knowledge of this disease has been found until the writings of Rufus of Ephesus regarding plague in Egypt in the second century B. C. At this time the disease was present in lower Egypt, spread along the coast of the Mediterranean and existed as a pandemic for an unknown period. Numerous detailed accounts give positive data that this pandemic was the same disease as that now known as bubonic plague. During this period war, drought and famines were prevalent throughout the world. During the centuries following this pandemic, numerous references to plagues are found, but each is too indefinite to warrant a diagnostication of bubonic plague.

In the Justinian age, beginning about 540 A. D., plague reappeared in pandemic form. Starting in lower Egypt, especially Pelusium, the disease spread along the shores of the Mediterranean to Asia Minor and Europe. In this pandemic plague existed in a given locality one, two or three years, disappeared to probably return at a later time. The nature of the disease was

similar in most respects to what we now know as plague. This pandemic continued for fifty or sixty years, and in all probability gradually died out. In this age wars, droughts and famines were also prevalent. During the middle ages numerous references to plagues are found, but again they are too indefinite to enable us to distinguish the nature of the disease. It is most probable that plague played an important rôle in all these epidemics.

In the fourteenth century plague again reappeared in pandemic form throughout the civilized world. This time the disease began in western Asia, traveled to Asia Minor, thence to Egypt, Europe and Russia. During this pandemic some places were infected only a few months, others for several years, while in others epidemics continued for one or two years, after which the disease disappeared to again reappear after a longer or shorter time. Again, war, droughts and famine were prevalent. Several centers, so to speak, became temporary distributing points for the disease. Of these, Egypt in Africa, Turkey in Europe, and Syria in Asia were the most important. Again in these countries the disease remained more or less active for three centuries, giving rise, from time to time, to epidemics in neighboring countries. As the centuries wore on, the number of epidemics gradually diminished until 1841, when the last epidemic of plague occurred in Constantinople. In recent times Arabia was visited by epidemics in 1853, 1874 and 1879, Tripoli in 1874, Persia in 1863 to 1878, Messapotamia from 1866 to 1876, and in Astrakhan in 1878.

From 1879 until 1894 the world was practically free from plague, but in the latter year an increased death rate among the Chinese at Hong Kong was observed, an investigation of which revealed the presence of bubonic plague, not only in Hong Kong, but also in Canton, where it probably came from Lientschau and Pakhoi. Since then the disease has spread to India, Japan, the Philippines, numerous ports along the route

to Europe, South America, South Africa, Portugal, England, San Francisco, Mexico and many other localities. We are, then, in the midst of another pandemic of bubonic plague. Thus plague has spread over the world in at least four pandemics. First, in the second century, second in the Justinian age, the third in the fourteenth, and the fourth in the nineteenth century.

A brief summary of the observations on these pandemics reveals the following:

As to Origin.—No mention is made regarding the origin of the pandemic in the second century B. C. In that of the Justinian age the disease began in lower Egypt to spread over the then known world. During this pandemic new infected centers of plague were established in western Asia, where the third pandemic in the fourteenth century arose. During the third pandemic new centers were established in southern China to give rise to the present pandemic in the neighborhood of Canton. Thus plague has shifted its home from Egypt to western Asia, and later to southeast China, remaining throughout the centuries in tropical and subtropical climates.

As to Infected Centers.—Probably owing to more accurate detailed records, plague has in each of the latter pandemics formed new temporary centers from which the disease spread over the neighboring countries. Of these, Constantinople in the fourteenth century, and Hong Kong and Bombay in the present pandemic are notable instances. In these centers the disease may occur from year to year or at irregular intervals in epidemic form, or its presence may be shown by occasional cases.

As to Routes Traveled.—During each pandemic the disease has followed the great paths of commerce. In the second century B. C. it spread along the coast of the Mediterranean; in the Justinian age, along the same routes to Asia Minor, thence to Europe and western Asia; in the fourteenth century, from western Asia to

Asia Minor, thence to Egypt, Europe and Russia, and in the nineteenth century from south China to Canton, to Hong Kong, thence throughout the world.

As to Classes Affected.—All races have been affected, especially the lower classes. Among the higher classes, living in the better districts of a city, the servants, as a rule, suffered. History abounds with instances of domestic and wild animals suffering from severe epidemics.

As to Modes of Dissemination.—Here records are liable to lead astray, for even now much mystery surrounds the dissemination of plague. It has been noted that clothing and articles of commerce have served to transport the disease, and the migration of rats, etc., has been observed.

Historical Recapitulation.—In each pandemic a new endemic home was established, first, in Egypt; second, in western Asia; third, in southeast China; temporary infected centers were established from which the disease spread in epidemic form; given localities were again and again infected; pandemics lasted from fifty to sixty years; in infected centers plague remained more or less active for several centuries, and the lower classes were mostly affected.

As the present pandemic first originated in Lientschau, then extended to Canton, from Canton to Hong Kong, thence to the civilized world, it is important to relate briefly the existing conditions in south China. The entire southern portion of China is thickly inhabited, with a large number of densely populated cities. Of these the most important, geographically and commercially, is Canton, which is located on the Pearl River about ninety miles northwest of Hong Kong, with which it is in intimate intercourse by water and land. It is a filthy, densely populated, poorly constructed city, consequently unhygienic conditions exist. Living under these conditions, the natives have a lowered resistance to infection of all kinds, consequently epidemic diseases

are most prevalent. On account of the filth and customs of the people, rats and insects are abundant. Similar conditions exist in all Chinese cities.

FEATURES OF THE BACILLUS.

The *Bacillus pestis,* discovered by Kitasato and Yersin in 1894, grows luxuriantly in all ordinary culture media, even under disadvantageous conditions. As our knowledge of this organism increases, its power to maintain life and virulence under unfavorable conditions becomes of more and more importance. It has been found that the organism lived nineteen days in the sunlight and twenty-three days in darkness, at ordinary room temperature; twenty-two days in a desiccator; eleven days in a thermostat; one hour in a temperature of 60 C., and fifteen minutes at 80 C. The German Commission found virulent organisms in sputum after ten days; Martini in sputum of a pneumonic case after seventy-six days; Abel in sterile water after twenty; Hankin in various cereals after thirteen; Jakote in buried plague cadavers after thirty, and in dried pulverized organisms of animals dead of plague after forty-eight. On dried paper kept in a thermostat, organisms have been recovered after forty-four days, and in direct sunlight, after from three to six hours. Under favorable conditions, as on culture media, the organism lives indefinitely. Its virulence, even in old cultures, is frequently as great as in young ones. The finding of bacilli which rapidly regained their virulence in cotton goods by Kitasato is one of the most important links in tracing plague from one country to another. When plague was discovered in Kobe, Japan, it was found that a large consignment of cotton goods had recently been received from Bombay. Two hundred tubes were inoculated with portions of these goods, from one of which a virulent plague culture was obtained. Several dead rats were also found in bales of cotton. It is more than probable that by this means plague was introduced into Japan.

These facts demonstrate the resistance of *Bacillus*

pestis to dryness and a moderate degree of heat for an undetermined period, and suffice to explain the origin of succeeding annual epidemics in a given locality, and to show one route and means of carrying the disease from one country to another.

The susceptibility of rats, mice, domestic animals, flies, flees, ants, etc., serves to preserve the life and virulence of the bacilli, and to spread them in accessible places. The migration of rats, etc., may serve to carry infection to neighboring districts.

As bacilli have been found in the blood during a period of forty-five days, and in sputum for seventy-six days, convalescent patients may be a means for carrying the disease from one district to another, or even to distant lands.

Returning to the origin of the present pandemic, it is more than probable that during the pandemic of the fourteenth century, or the numerous epidemics of the following three centuries, south China was visited by plague when a new endemic home was established. After an indefinite time the disease increased, extending by ordinary routes of travel, in merchandise, etc., from outlying districts to Canton, where, favored by the filthy, crowded condition of the city, an extensive epidemic immediately developed. Here merchandise of all kinds comes in intimate contact with infectious material, and undoubtedly harbors more or less bacilli. Hong Kong, the port of Canton, undoubtedly received its infection from Canton by means of merchandise, individuals who might have contracted the disease in Canton and developed it in Hong Kong, or possibly by means of rats boarding steamers at Canton.

Hong Kong is one of the most important shipping centers in the Orient. Here practically all vessels plying in Oriental waters touch for coal, cargo and the transshipment of cargo, all of which are handled by Chinese coolies who live in the plague districts of the city. In

addition to cargo, rats in large numbers often board the steamers to be transported to distant ports.

In the spring of 1894 plague spread from Canton to Hong Kong, thence to a number of coast towns in southern China, and later to many distant ports, or in other words, the disease followed the ordinary routes of travel.

In the present pandemic Canton and Hong Kong are the chief distributing centers for the world, thus history is repeating itself. Consequently we may anticipate the presence of plague in these cities for an indefinite period to give rise to epidemics of plague at irregular intervals in various portions of the world.

TRANSMISSION OF PLAGUE.

Transmission of plague is best considered under the headings of portals of entry, direct and indirect transmission.

Portals of Entry.—On account of the absence of local reaction in the great percentage of cases much doubt exists as to the real portal of entry. This is made more prominent by the slight local reaction found in the small number of cases in which a primary sore is found. Here almost no change in the surrounding tissues are noted, frequently only a slight lymphangitis is observed, and usually the first lymphatic gland draining the infected area is the primary bubo. The absence of a slight local reaction about a primary sore leads one to think that a small wound in the skin might be infected without visible local reaction.

Experimentation on lower animals shows about the inoculated area an intense edema, more or less hemorrhage, and considerable infiltration, with the lymphatic glands draining this area enlarged, hyperemic, and often hemorrhagic. It must be remembered that in experimentation a very large number of bacilli must be inoculated. As in all of these experiments lymphatic glands draining the inoculated area are the first affected, it seems most reasonable to suppose that primary buboes in human beings indicate the region through which the

infection was received. In the unshod Oriental races about 60 per cent. of primary buboes are femoral or inguinal, 25 per cent. axillary and the remaining cervical. These percentages fairly accurately represent the relative exposure of the several regions to infectious material. The relative frequency of primary buboes in children strengthens this supposition. Here the largest percentage is found in the cervical regions, next in the axillary and inguinal, and femoral least of all. It is well known that whenever possible a child will put everything into its mouth, and is inclined to play on the ground, where the hands are more liable to infection than the feet. As the larger percentage of cases are of the bubonic type, it seems most reasonable to suppose infection is received through superficial wounds in the skin and mucous membranes of the mouth and nose.

The apparent immunity of oil workers and those well clothed seems to favor the opinion that infection is most often received through the skin.

Wilms thinks the digestive tract is an important portal of entry, but numerous observers doubt its importance. In two epidemics I saw no evidence of infection through this tract. Experimentally primary mesenteric buboes have been observed.

Infection through the lungs undoubtedly occurs, especially in pneumonic cases. Deane considers the pulmonary tract the most important portal. If this were true, it would be as difficult to explain the absence of local pulmonary changes in the great percentage of cases as it is to account for the lack of local reaction in skin wounds. Again, it would be difficult to explain the distribution of primary buboes if the bacilli entered through the lungs.

Some evidence points to the sexual organs as a portal, but more observation is needed to establish this fact.

Direct Transmission.—Here the most important feature is the infected human body. As septicemia develops, all organs, secretions and excretions become infected.

Of especial importance are the urine, feces and sputum. In pneumonic cases the sputum is ladened with bacilli, and in delirious cases is a dangerous source of infection, as the bacilli may be spread broadcast about the room and over those in attendance. Here the resistance of the organism to dryness is of great importance.

Owing to hemorrhages in the stomach cases in which vomiting is present are liable to infect the surroundings. Pus from buboes and serum from primary sores, etc., are also infectious. The cadaver is teeming with organisms, consequently is a source of infection until destroyed.

Naturally, all attendants are continually exposed to these various sources of infection, and it is astonishing that so few of them contract the disease. Perhaps the constant exposure develops a sense of precaution carried out in the minutest details. Many instances of direct transmission of plague from lower animals to man are on record, but generally speaking, direct transmission plays an unimportant rôle in the dissemination of plague; as an evidence, the small number of attendants in plague hospitals contracting the disease may be cited.

Indirect Transmission.—Here the degree of viability and virulence of the organism is an essential feature. As the *Bacillus pestis* is a long-lived organism, and as the excreta, etc., are ladened with bacilli, all material coming in contact with the sick or kept in houses in which patients have been kept may serve to harbor and transport infectious material to those handling these goods.

Dust arising from infected places may be infectious for an undetermined time, certainly much longer than was formerly supposed. No positive evidence to show that water may transmit the disease is at hand. Perhaps the most fruitful sources of infection are the dark, poorly ventilated, filthy houses inhabited by the lower classes. There the organisms might easily remain alive

and virulent over long periods, to be picked up by the unshod or otherwise exposed inmates.

Perhaps the best evidence of the importance of indirect transmission is the fact that those well protected by good clothing and shoes and living in fairly good hygienic condition rarely contract the disease.

In conclusion, it may be said that plague sprang from Egypt, formed new endemic homes, and traveled along the routes of commerce. The *Bacillus pestis* is long-lived, and may be carried from place to place in merchandise, by rats, etc., and by convalescents; the disease thrives best in unhygienic places, and the lower classes are mostly affected.

THE PROPHYLAXIS OF PLAGUE.

J. J. KINYOUN, M.D., PH.D.
GLENOLDEN, PA.

The *sine qua non* in plague prophylaxis is the diagnosis. This is not so easy a matter as some are led to believe. The many forms which plague infection may assume, taken together with its insidious character, the inexperience of the medical profession of a given place, may render its recognition extremely difficult, even notwithstanding the fact that accurate methods of examination can, with certainty, be relied on to detect it.

The chief difficulty, however, which confronts one is that it may be so easily diagnosed as one of certain diseases of local origin, which it may so closely resemble, that it is an easy matter to overlook it. If the first cases were of the frank type—the bubonic—the task would be made easy. As a rule, this does not occur, and the ease in recognizing them as advocated by some is more a theory than a condition.

The clinical manifestations of plague are almost as varied as those attributed to malaria. Cases may resemble in whole or in part, and have been confounded with anthrax, pneumonia, diphtheria, bronchitis, pleurisy, pericarditis, endocarditis, peritonitis, appendicitis, rheumatic, typhoid, typhus, relapsing and malarial fevers, miliary tubercle, dysentery, beri-beri, syphilis, venereal bubo, mumps, septicemia and adenitis.

While it goes without saying that if it were the custom or the rule to apply accurate examinations to all cases in the foregoing list, nearly, if not all, the first

cases would be detected, but we know that there are so many obstacles in the way for such procedures that it would be impracticable to carry them out, even if the medical profession were willing or able to do so.

If, on the contrary, all the first cases of plague infection were to be found in well-appointed hospitals, where accurate observations could be carried out, these would give us little concern. But this does not obtain. The first cases, as a rule, do not go to hospitals; in fact, only a few filter in, the greater number being found in localities where one is least expecting them and usually among a class of persons who naturally oppose the unwarranted (?) and presumptuous interference of the health authorities, or the physician who may have suspicions. Many cases occur under these conditions, and are not seen by anyone before the services of an undertaker or coroner are required. It is remarkable how resourceful this class of persons is in attesting to the non-infectious nature of the malady which has carried off their relative and friend.

To give a few illustrative experiences on the difficulties of a diagnosis: A case of illness occurred in San Francisco which, clinically, was that of typhoid fever, and passed muster as such, and three weeks later the autopsy and bacteriologic findings were those of plague. Another case was clinically that of diphtheria, but no cultures were made from the throat; antitoxin was administered when the case was moribund. During his last hours, while in delirium, he coughed and spat in the nurse's face, some of the sputum entering the eye. The nurse was immediately immunized with a large dose of diphtheria antitoxin. Despite this precaution she became ill within less than 30 hours, and died of an acute fever four days later. Autopsy revealed an acute septicemia, due to plague. Another gave a typical clinical history of lobar pneumonia, and the death certificate was made out accordingly. The room in which the patient died was closed for two weeks, when it was occu-

pied by a woman and child. Four or five days after occupancy both became ill, one with bubonic, the other with pneumonic plague. A case was diagnosed as phlegmonous erysipelas, and was treated as such for 10 days, but on postmortem examination plague bacilli were isolated from the phlegmonous tissues, the heart's blood and spleen. Another case occurring soon after certainly did present the evidences of acute myocarditis. The blood, however, showed leucocytosis and pest-like bacilli. The autopsy was confirmatory of plague septicemia.

In Hong Kong I saw in consultation a case which all of us agreed was one of acute appendicitis, requiring immediate surgical interference. The blood showed considerable increase in the number of white blood cells, and many malarial parasites. The surgeon concluded to wait until quinin had been administered. On the next day the patient was worse, temperature higher, slight effusion into the peritoneum. The operation was deferred, and death occurred on the next day. Autopsy revealed a plague infection of the retroperitoneal glands near the appendix. In a case of a child in Manila presenting all the symptoms of a catarrhal pneumonia, autopsy confirmatory, the bacteriologic examination showed the cause to be plague bacillus.

The macroscopic appearances of organs and tissues of pest infections are, as a rule, quite distinctive. There is a respectable number of these in which there is scarcely anything suggestive of plague infection; yet the bacteriologic examination will reveal pest bacilli in all. Mixed infections may also be misleading, as instanced in the case diagnosed as appendicitis and in cases of beri-beri with an intercurrent infection of plague.

A vessel arrived at one of our quarantine stations with many cases of beri-beri aboard. Several had died en route or soon after arrival. Specimens of organs removed from a case just dead were examined bacteriologically and the pest bacillus isolated. The macro-

scopic, as well as the microscopic, appearances of the tissues were typical of those of beri-beri.

The lesions observed in a respectable number of plague cadavers, both in San Francisco and in Hong Kong, were about as varied as one could wish to see. Those in Hong Kong occurred at the time of year which might be termed, for want of a better name, the post-epidemic period when the disease was declining; that is to say, when the number of cases were fewest. In these, the frank bubonic type was of rare occurrence, the septicemic type preponderating. Probably many of these do occur during the time when the epidemic is at its height and are overlooked. These cases certainly do occur in the pre-epidemic period and are not recognized as plague. Some of these are mild, ending in recovery; others are disposed of under a mistaken diagnosis. Viewed from a public health standpoint, these cases are of the greatest importance, and are, it is believed, one of the chief sources perpetuating the infection. These are some of the obstacles which stand in the way of a correct diagnosis, and are mentioned simply to emphasize the importance of making a careful and painstaking examination of every case wherein there is a possibility of plague infection. If such could be done, the majority of these masked cases would be recognized, yet some will be overlooked, even after the most painstaking examination, because of our inability to isolate the organism *intra vitam* or postmortem.

Plague, like cholera or bacillary dysentery, may show the same variations of virulence. It may be at first slight, and require a time more or less long before it assumes a virulence sufficient to cause an epidemic. On the other hand, it may remain for a long time in a locality making but little progress. Climatic conditions play no small part in its epidemicity. In such places as Hong Kong, Canton, Amoy and Manila it is certainly influenced by the temperature and humidity. The disease begins to assume its epidemic character during the

warm rainy season, gradually increasing in intensity until the end, when it declines. It has been noted that when the temperature reaches 80 F. and is constant it declines gradually, reaching its lowest ebb just before the rainy season begins again.

While this condition obtains in the aforementioned places, it shows quite the opposite in California where the greatest number of cases have occurred during the dry season. Seasonal influences appear, then, to have also something to do with the type of the disease, particularly in Hong Kong where during the rainy season the majority of cases are bubonic; in the dry season, when the temperature is high and constant, nearly all the cases are septicemic. Just why there are these variations in type, or frequency of infection, can not be explained.

The ambulant type is not so much a factor in the dissemination of plague as some have been led to believe. The experiences of the health authorities of Hong Kong, Manila and at the Japanese ports all agree that these cases are extremely rare. My own experience, while not so extensive as that of others, is confirmatory. During 1899 to 1901, in over 50,000 persons examined for the apprehension of just such cases, out of these not more than 6 suspects were found—two on a vessel undoubtedly infected. These were kept under surveillance and a most rigid examination made of them; yet, notwithstanding this, the cause was not found to be other than plague.

The disease is a filthy one, and, like water, seeks its level among conditions which are ideal for its propagation and dissemination. Some years ago I made a statement that so long as the Chinese exclusion act was in force there would be little or no danger of the introduction of plague from the Orient. Since then I have learned that the Chinese exclusion act does not exclude, in the strict sense of the word, and particularly so from a health standpoint. There is a constant interchange

going on of the Chinese coolie population between China, the United States and its territories, this interchange being provided for by special regulations. There also must be considered a number of persons who are debarred under the provisions of the act who seek admission through other channels not contemplated or specifically provided for. There can be no question that a respectable number of such persons do gain entrance to the United States. Another feature in the trans-Pacific traffic is the increasing importations of commodities from the Orient, which must be given due weight as a factor in the transmission of plague. It is farthest from our purpose to cast undue reflections on our own people, but there are certain conditions at present existing and constantly increasing which can not be viewed without concern.

There are many localities in our country which by reason of climatic conditions and race habit are ideal for the propagation of plague. The constant influx of foreign populations of low standard gravitating to many of our urban centers, is gradually forming the nucleus of material for epidemics, and is by reason of this influx becoming a serious problem in our public health work. With regard to the Chinese population, it is without doubt the worst of all as regards plague. There are now about 120,000 Chinese located in the United States, the majority of whom are on the western coast. These are controlled, and I may say absolutely, by that great and powerful organization known as the Chinese Benevolent Association, or The Six Companies. The rule of this organization over its subjects is as autocratic as the Sultan of Morocco or the Shah of Persia. The Chinese of the eastern coast are controlled by another organization of similar nature which is no less autocratic. These two instances are cited to show the manner of controlling the Chinese, and because bubonic plague has appeared among this people, and for offering an explanation in part of the

difficulties which have stood in the way of its eradication among them. Some observers believe that little or no progress will be made in the detection of cases of plague among the Chinese, if a postmortem examination is insisted on, because of the strong race prejudice against them, as it will tend to the concealment of cases and more harm will follow than good accomplished. From our own observations this is only partly true. It is not so much due to prejudice of the Chinese against the examinations so much as their fear of their tong, or tribal customs and government being interfered with by western ideas. The Chinese desire to be let alone. The depositing of their dead on the streets, the concealment of cases, or the surreptitious removal of the moribund to the "Halls of Tranquility" is not so much their fear of a desecration of the dead as that of interference with their commercial and social life by the sanitary authorities. A careful examination of all sick of a Chinese community where plague is once detected, and a postmortem examination of all dead is the only way in which all plague cases will be found.

MEASURES TO BE DIRECTED TO THE INDIVIDUAL SUSPECTED OF BEING AFFECTED WITH THE PLAGUE.

The suspected case should be isolated. The isolation should be all the term implies. Pending the completion of a diagnosis the same care should be exercised as if it were proven to be plague, and as soon as the examination is complete it should be removed to a hospital provided for infectious diseases, where the dangers of infection can be best controlled and where the case can be medically and humanely treated.

The premises in which the case is discovered should be regarded as infected—doubly so if it can not be definitely proven that the infection was contracted elsewhere. The house should be evacuated and be immediately closed. Sometimes, however, the condition of the case is such that it can not be removed; if so, it may

be necessary to assume an extra risk and convert the premises into a temporary hospital. As soon as the premises are ready to be evacuated provisions should be made for not only the care and treatment of those sick, but of those who have been in contact with it. These should be held under observation in a non-infected place for a sufficient time to demonstrate their freedom from infection.

The premises and contents should be disinfected immediately after their evacuation by persons who are thoroughly conversant with the work, by the following methods: (*a*) by gaseous disinfection; (*b*) by mechanical cleansing; (*c*) by chemical disinfectants; (*d*) by steam; (*e*) by burning.

Great care should be exercised in the application of measures for the destruction of vermin, not only in the house itself, but in the immediate vicinity. If rats or mice are found dead in these places a bacteriologic examination should be made of these places to determine whether they are infected, as much depends on the thoroughness in which this feature is carried out.

After completion of the disinfection the house should be thoroughly aired. The soil in and around the premises may be such as not to exclude the possibility of its containing the plague bacilli, and if such be the case, it should, of course, be subjected to such processes as would sterilize the surface at least.

Suspects may be divided into two classes: 1. Those coming in direct contact with the case. 2. Those who have not come in contact with the case, but who have lived in the same house or in close proximity thereto. These may or may not be in the incubative stage. If so, the advisability of administering prophylactic injections of anti-pest serum must be considered, or perhaps the protective inoculations of Haffkine.

Too much importance can not be given to the destruction of vermin, and should not in any case be confined to the premises or those contiguous thereto, but should

include a careful search for sick or dead vermin in a considerable zone around the infected premises, together with the destruction of all vermin. The crusade against vermin should be conducted for a sufficient length of time to be doubly sure the danger of their infection is past. This does not mean a few weeks, but should be persistent for months, or even a year or more after the last case of human plague.

The crusade against the rat population in San Francisco during the past winter demonstrates that it is infected. Just to what extent it is not safe to say, as the number examined is entirely too small to base an opinion on, but so far it has shown a greater number of infections than has been observed in Japan; in fact, nearly as large as in Hong Kong. It is, therefore, not safe to say that because no pest rats are found for two or three months the epidemic among rats has disappeared.

There are many rats in San Francisco. Judging from my own observations, together with those of two of my colleagues, the number is beyond computation, especially so along the water front. In the Chinese quarter, fortunately, there are fewer rats. Possibly this may partly explain why there has been so few cases of plague, but even if it were so it does not lessen the danger of the rats becoming infected along the water front. It is safe, therefore, to state that the rat population of San Francisco is stationary, as the few destroyed is a mere bagetelle. What should be done is to institute a systematic and thorough campaign against the rat, which should take in the whole city.

MARITIME COMMERCE IN ITS RELATION TO THE SPREAD OF PLAGUE.

Plague, like cholera, follows in the wake of commerce. For a long time many features connected with the transmission of plague by vessels were not understood. While it was easy to discern the relation existing between a vessel having cases of plague on board and others contracted from these on land, we were often

at a loss to discover the origin of outbreaks occurring at seaports which could not be ascribed to vessels or their personnel. The various theories in vogue from time to time were a natural sequence. The careful observations of the past few years have been of greatest value in determining just what part the vessel, cargo and personnel play in the transmission of plague. Is it possible for the personnel of a vessel to bring the infection aboard, either in their persons or in their effects? Only a few instances have been cited where the personal effects of the personnel have been responsible for plague appearing on shipboard. Ruffer believes that a few cases are traceable to this cause. The dirty, filthy rags of the Indian pilgrim coming from Bombay and Calcutta were thought to be responsible for the outbreaks on shipboard, yet these were few and could not be traced directly to these as the cause *per se*. Kitasato mentions a box of clothing of a soldier who died of plague in Formosa sent to Kobe, and soon after opening it plague attacked some of those who had handled these articles.

It would perhaps further our understanding of this feature if a few details were indulged in which relate to one of the chief sources of infection to this country. Vessels in Hong Kong harbor, as a rule, lie at anchor some distance from shore, communication being held by steam launches, the native sampan and cargo boat. Very few of the trans-Pacific vessels ever touch at a dock. Nearly all these carry a full complement of Chinese for their crews. These are shipped in Hong Kong for the round trip and are changed on each return voyage. Only a few of the crew come from Hong Kong, the greater proportion being drawn from Canton and its immediate vicinity. As soon as a vessel arrives in Hong Kong a crew is discharged, the members return to their homes, and if any of these reship they return only a day or so before the vessel sails. These, together with the new members, constitute a fresh crew.

Early in 1899 cases of plague began to appear in several of these vessels, usually from three to five days after leaving Hong Kong. These were apprehended at the Japanese quarantines. The short time elapsing between the time of departure and the appearance of the cases pointed strongly to the fact that these were either pestis minor or pestis major in the incubative stage, and presumably the latter, as all attacked died. The possibility of the infection being brought on shipboard in their personal effects can not altogether be excluded, yet the weight of evidence leans further to the side of the person being the factor, rather than the effects.

While serving as quarantine officer at the port of San Francisco I soon became convinced that there was great danger from this source, and in response to persistent and urgent appeals, the surgeon-general finally detailed officers of the Marine-Hospital Service for duty at Hong Kong to make the inspections and to exercise a sanitary surveillance over all vessels destined for the United States. Since this inspection has been established, the effects of the crew and steerage passengers being disinfected, no case of plague has occurred on any of these vessels. Many cases of incipient fever, etc., have been excluded, and some of these have died soon after with plague. This would seem to indicate that the cases which had heretofore occurred on these same vessels were in reality suffering from plague on departure.

The Canadian vessels, for purposes and reasons best known to the officers of that company, have adopted a policy of inspection only, claiming in so doing that the restrictions imposed by the United States were worse than useless; that their system of inspection without disinfection was far better or "just as good." It almost seemed so until a case of plague was detected on the vessel's arrival at Nagasaki. Possibly this case was due to infection from some article taken on board, but it is also more than probable that on account of the short time elapsing before the appearance of disease (4½

days) that the personal inspection was at fault—a case of plague in the incubative stage was allowed to go on board.

The sanitary history of these vessels is quite instructive. Seven vessels had plague on board en route from Hong Kong to San Francisco; in only two did secondary cases occur. In five vessels cases occurred soon after leaving Hong Kong; in one of the two about 10 days after leaving port, in the other over 14 days. From a careful inquiry it seems to establish the fact that these cases occurring as soon as four days from port were in the incubative stage, whereas those developing after 10 to 14 days contracted the infection through some agency on shipboard.

CARGO.

Since the days of the Levantine Lazaretto, the cargoes of vessels have been regarded with suspicion, and a belief held that such were capable of transmitting contagion. Certain articles of cargo are no doubt the means of preserving and transmitting the contagion of certain diseases, and are also capable of being infected, particularly so on shipboard. It has never been accurately determined how plague reached Bombay from Hong Hong. All the evidence points to an infected vessel. Certainly if a vessel can come from Bombay to Yokohama with plague-infected rats on board, or a vessel from Bombay arriving at Kelung, Formosa, with a case of plague, we would reason that it would be equally possible for infection to reach Bombay from Hong Kong.

From all information obtained, the cargo is not believed to be the principal means of its dissemination of plague. The cargo in most instances is free of infection at the time of placing on board, yet it may be infectible by agencies on board. There is a suspicion that certain food products from China may, either through their mode of preparation or storage, become infected

with the plague bacillus, but these instances are rare. Cargoes of grain, particularly rice and of forage, may become infected by the same agencies. It is more than a probability that the disease was carried to Oporto, South Africa, New Caledonia and Calcutta in such cargoes. Kitasato's researches also appear to establish the fact that infected cotton shipped from Bombay to Osaka was the means of introducing plague in the latter place.

We can not, however, rest the case on the cargo alone as being the means of conveying plague, and must not be unmindful of the close relationship existing between the rodent and plague on land. The same will also apply to vessels. While certain classes of food products and other kinds of cargo may be infected with plague, I think all will agree that it is of minor consideration as compared to the dangers arising from plague infected rats on shipboard. These may become infected either by persons or their effects, or from infected cargo. On the other hand, they may become the means of infecting cargo. The steamship *Friary* arrived at Hull, England, about 80 days out from Montevideo. Two cases of fatal illness occurred en route and soon after departure; all were well on arrival. A few days after cargo was broken and begun to be discharged by the crew, when soon thereafter two became ill with symptoms of influenza, and were sent to the hospital. One of these died shortly after. About this time several mummified rats were discovered among the cargo. This, taken together with several other cases, led to a more careful examination, which demonstrated the cases were not influenza, but plague. The inquiry further developed that all the cases of plague had come from direct contact with the dead rats.

Another steamer arrived at Hamburg from Rosario, Brazil, where plague was prevailing. All were well during the voyage and on arrival. The quarantine inspection revealed several dead and sick rats. These were examined

and found infected with plague. Another instance was an infection of the personnel by rats in the case of the steamship *Coptic,* which sailed from San Francisco for Asiatic ports in August, 1900, and 21 days later, just before arriving at Yokohama, a case of plague developed in a passenger. On clearing out the compartment two dead plague rats were found. Another steamship plying between the United States and Asiatic ports demonstrates the fact of rat infection perhaps in a more remarkable manner than any of the former.

A vessel left San Francisco early in the spring of 1901 for Asiatic ports, making the round trip in about four months; the vessel did not touch at any wharf, but discharged passengers and cargo well out in the stream. On her return voyage, a few days out from Honolulu, two deaths occurred between Honolulu and her home port. There being no physician aboard, nothing more definite than the master's statement could be obtained, but all pointed to plague. On discharge of the cargo no dead rats were found, and no information could be obtained regarding them from any member of the crew until finally the Chinese quartermaster admitted that dead rats were being found continually when the vessel lay alongside the dock in San Francisco, and were observed on several occasions on the outward voyage. Here was an instance where rats were dying as long as three months before any case occurred among the crew, notwithstanding these must have been in close proximity to the personnel. Other instances of the same character could be cited if it were deemed necessary.

I can not, therefore, bring myself to agree with the opinion of the British Plague Commission when it says: "There is no evidence that plague-infected rats have ever carried plague from one country to another, and that fumigation of holds of ships, in order to kill rats, is unnecessary and impracticable," or that there "is no confirmation of the suggestion that plague may be conveyed on board by embarkation of plague-infected rats."

It is indeed a fortunate circumstance that few of the vessels engaged in the trans-Pacific trade touch at any wharf in the Orient, and that passengers and cargo reach the vessels by boat. If it were otherwise it would be next to impossible to prevent these vessels from being infected on nearly every voyage.

Notwithstanding the present arrangement, the dissemination of plague has been very closely connected with some of these, and this is due more to their being infected with rats than to any other cause. Few rats, if any, get aboard these at Hong Kong, or at any other port, still these vessels contain numerous rats. It has been the custom since medical inspectors have been on duty at the Oriental ports to destroy the rats on board just after discharge of cargo by fumigation, but on the next voyage about the same number are on board. The question naturally arises, from whence do these rats come? The answer to this was the same from all, "American ports, principally San Francisco." The vessel is, therefore, subjected to a double danger from (*a*) cargo possibly infected, (*b*) and from infected rats.

With regard to the possibility of vessels becoming infected with rats at San Francisco, I may add my personal observations to that of the officers of vessels, whose answer to the query is quoted in the above. The wharves and water front are infested with rats in great numbers. The wharves are, as a rule, old, and of such construction as to be ideal vermin harbors.

These are some of the reasons explanatory of the way in which plague may be carried from place to place by means of vessels. A rat infection may exist comparatively for a long time before it is manifest, but just the same as on land. Places may become infected one after another by such vessels, and the circumstances attending its sanitary history be forgotten before the place awakes to the realization that it has plague.

MARITIME QUARANTINE.

On arrival of a vessel from an infected port great

care should be exercised in making the examination, and a diligent inquiry should be instituted regarding all that is essential in determining the sanitary condition of the personnel, cargo and vessel. The sanitary authorities should be given wide latitude in their determination of the fitness or unfitness of a vessel to be passed or to be held for treatment. The regulations for their guidance should be laid down on the broad principles of preventive medicine and not "line on line and precept on precept" of laboratory technic and unimportant details.

A few good rules are excellent, but rules enacted into law are still better, for regulations are not laws and can not have the force of law. The sanitary authorities should be experienced men, well qualified, and, above all, resourceful and possessing executive ability, and, moreover, endowed with a liberal amount of common sense. If these precepts are regarded, I feel sure that many of the hardships, both to commerce and the personnel of vessels, would be, in a great measure, removed, and the health interests of our country be better conserved.

In general, the same rules of procedure as suggested for cases on land are applicable for the treatment of a vessel, its cargo and personnel: The diagnosis, treatment of cases, surveillance of the contacts and the suspects, each with its appropriate treatment, and lastly, the disinfection of the vessel and cargo with the destruction of vermin.

The maritime quarantine stations at our larger ports are now equipped with all the requisite apparatus and conveniences for the treatment of such vessels with effectiveness and dispatch. A description of such stations will be unnecessary. One of the greatest safeguards to this country in preventing the introduction of plague is the present policy of the national government in detailing officers of the Public Health and Marine-Hospital Service for duty in the office of the consulates in the several Oriental ports. No one thing has

been of greater protection than this, as it has prevented plague from being brought on board, and, moreover, practically relieved commerce of many of the vexatious restrictions which were necessary to impose before this was done.

With regard to the utility of the detailing of medical officers of the government in European ports, it is a question whether any great benefit would be derived from it. The conditions under which a sanitary supervision of vessels and their personnel would be maintained, would amount to but little. As a public health measure, it has practically not been appreciated by the trans-Atlantic traffic, and what little has been allowed to be done rests more on the fear of a quarantine on this side rather than being one of co-operation to prevent infectious diseases being brought to this country.

MEASURES TO BE ENFORCED IN A PLACE INFECTED WITH PLAGUE.

As before stated, the prompt recognition of the first cases is most essential. A single case, for example, coming from a vessel or another place on land, if promptly recognized, would give us but little concern. But such instances are rare. It is usually not recognized, and one is not apprised of the fact until weeks, or perhaps months, afterward.

Plague is believed to have been introduced into Hong Kong from Canton at least eight or nine months before the cases were of sufficient number to attract attention by the increased mortality from "fever." Therefore, when plague is recognized as plague the initial focus usually has been lost. Further, it may, and perhaps is, in the majority of instances, introduced by rats, rather than by persons and their effects. This was true of the first and second epidemics in the Hawaiian Islands. Rats were observed to be dying in and around the wharves many weeks before the first cases appeared among the Chinese and native population. The last epidemic was directly traced to infected rats.

All rat plagues are not followed in direct sequence by human cases. These may exist for a long time and not be infective. On the other hand, they may occur just slightly before or be synchronously with the plague in man. Davis says: "Plague, when it is introduced by persons, may be readily exterminated; when introduced by rats, followed by the infection of shore rats, it has come to stay."

During the first great outbreak in Hong Kong, in 1895, rats were found dead in many of the houses occupied by the European population; yet but few of these houses became infected. In contrast with this, in 1901 the majority of the Europeans affected with plague was evidently due to infected rats, as these were found in nearly every instance. Nearly as late as 1900 the medical officer of health says: "It is also interesting to record that, although over 400 Chinese coolies have been employed regularly in scavengering and cleansing the city and in the disinfection of infected premises, and that practically the whole of the 42,000 dead rats must have been handled by these men, yet only three cases of bubonic fever are known to have occurred among them during the year." "In 1901," he says, "there were about thirty men who made a business of collecting these rats, and I find that no less than seven of them died of bubonic fever during the period under report, while five others left the colony because they were sick, and two of these later are said to have died of this disease on the mainland."

In Kobe plague first made its appearance along the water front just close by the place where the cargo junks were moored. The first recorded case, however, was not among those who lived on the boats, or, from what could be learned, had anything to do with the loading or unloading of vessels. It may have reached the shore through the agency of these junks, but, what is more probable, by a wharf at which vessels arriving from Kelung, Formosa, Hong Kong and Bombay dis-

charged their cargoes. As soon as plague was recognized the work of its eradication was placed in the hands of Professor Kitasato, to whom all praise should be given for his great and noteworthy achievement in freeing Japan from plague.

Japan has so far been the only country which has successfully eradicated plague within a short time. The system adopted by the local and imperial authorities, while in some respects a rigorous one, has demonstrated that it is the only kind which is efficient. As Dr. Thompson has remarked: "There is no royal road to the prevention of plague." It means work, and hard work at that. Although much information has already been given in the published reports of the plague epidemics of Kobe and Osaka, there are many features and details of these which have not been set forth as clearly as they should be, and, therefore, are deemed of sufficient importance to describe some of these more fully, with the hope that a better understanding of the subject of plague prevention and eradication in Japan may be of service in our country, particularly in dealing with populations and conditions which may be similar.

In 1899 cases of plague were discovered among the population of Kobe which lived along the water front and contiguous to that part of the bay used by the cargo junks. Soon after the announcement of the discovery of the first cases, the imperial government sent a commission of experts headed by Professor Kitasato, which was empowered to co-operate with the local health authorities, and to take such action and adopt such measures as were deemed necessary to eradicate the disease. Immediately on assuming charge, an inspecting corps of thirty-six physicians was organized to make a thorough inspection of the city. Volunteers were called for, and as many as 380 physicians were selected and appointed for this work. These were divided into the following classes:

1. Inspectors for districts, whose duties were to see all

cases of illness, it mattered not what the nature might be, to view and obtain the history of all those dead and report the same to the district superintendent.

2. A corps of inspectors for performing autopsies and making bacteriologic examinations of all persons dead suspected of plague.

3. A corps of inspectors whose sole duties were to make bacteriologic examinations of rats.

4. A corps of physicians to administer the anti-plague serum and Haffkine-Shiga prophylactic.

5. A hospital and ambulance corps to look after those sick with plague and those detained in quarantine.

6. A disinfecting corps, organized much in the same manner as a volunteer fire department.

7. A corps of rat inspectors detailed from the police force.

The plague made considerable progress in Kobe before the authorities were able to cope with it. It was decided from the first that all houses in which plague had occurred, or those contiguous thereto, should be destroyed, but the authorities were immediately confronted with a law which prevented the destruction of buildings by fire. It took some few weeks before this law could be modified, and so valuable time was lost. However, as soon as the law became operative, the occupants, with their contents, were removed under sanitary conditions, the houses razed to the ground, carried out by piecemeal and burned. In view of the success which the Honolulu authorities had in the dealing with plague, it was believed that this drastic measure would be all that would be required for the control of the disease. This, however, did not suffice. It is believed the reason why it failed was simply because of the interval between the time that plague was first discovered and the law went into effect authorizing the destruction of buildings.

Soon thereafter plague appeared in the central parts of Kobe and the adjacent municipality of Hiogo. Many

of the houses in which plague had occurred were disinfected and allowed to remain closed for ten days before the occupants were allowed to return. In several of these houses plague again broke out. Professor Kitasato was at a loss to explain just what was the reason, because a most thorough surface disinfection had been made of the houses and surroundings. When these cases occurred another sanitary inspection was made and several dead rats were found in the walls and over the ceilings of the houses. These rats were in various stages of decomposition, some evidently having been dead for several weeks. A bacteriologic examination was made of all these, and in a few only the plague bacillus was isolated. A vigorous crusade was then commenced against the rat. In fact, it was contemplated to destroy all the rats in these two cities, and a premium of 5 sen (2½ cents) was offered for every rat brought to the police department, either dead or alive. Arrangements were made that those bringing rats should report to the nearest police station, where a full history of each rat was obtained. This plan was not successful—only a few rats were brought by the population, or caught by those specially designated for this duty. The superintendent of police of Kobe hit on a plan which was one of the most successful features of the anti-rat campaign.

It appears that several years ago the government interdicted gambling in all its forms in Japan. Now it is a well-established fact that wherever you find the Tartar blood you will find also an inherent desire to play at games of chance. For all these years the Japanese nation had been interdicted from gambling, and they were practically in a state of hunger for hazard; so the superintendent of police suggested that in view of the fact that as the people were desirous to play at games of chance, in addition to the price of 5 sen offered for each rat, the lottery feature would be added, and a cash prize would be awarded at stated intervals. Each

person who would bring in ten rats was entitled to a lottery ticket and the prizes offered were: 100 yen, first prize; 50 yen, second prize; 20 yen, third prize; 10 yen, fourth prize; 5 yen, fifth prize; 1 yen, sixth prize.

As soon as this feature was announced, the number of rats brought in immediately arose from 1,000 per day to between 5,000 and 6,000 per day. So fashionable had rat catching become that people from the surrounding towns brought dead rats as a visitory offering to their friends in the city.

You can well imagine that the bacteriologists who examined rats were kept hard at work to examine from 5,000 to 6,000 rats per day. The number of rats that one physician could examine, a report was made, was not more than 200. This rat crusade developed some very interesting features. One was that in Kobe and Hiogo there was an intimate association between the rat cases and the human cases. Many times, however, the rat infection was not followed by human cases, and a few human cases were found in which there could be no connection traced between these and pest rats.

A short time after the epidemic of Kobe, plague appeared in Osaka. Osaka is the great manufacturing city of Japan, having a population of about 1,000,000. It is located about eighteen miles from Kobe, and is easy of access either by land or sea. In the city of Osaka, by reason of its location, the conditions were quite different from those in Kobe. The latter is a small, compact city, situated in a narrow strip of land on the sea, surrounded on three sides by hills; it is well drained and has a good water supply. Osaka, on the contrary, is spread over a large flat plain, intersected in every direction by canals. The intercommunication being very largely by water, the population was an ideal one in which an epidemic disease like plague would spread. Plague broke out among some of the workers of the large spinning mills, and it is believed now that some of the infection

can be traced to plague-infected cotton brought from Bombay. However, be that as it may, the authorities immediately saw that they had a very much more difficult proposition than that of Kobe. In the first place, the commercial and manufacturing elements protested strenuously against any such drastic measures which were enforced in Kobe, and so great was their influence that there was, for the time being, a modification of the measures which had proven so successful in Kobe, and they were suspended and the former status re-established. The rat examination, however, went on as usual, and it was not long before pest rats were found in several parts of the city, and at places quite distant from the place where the first cases were discovered. Again sanitary measures were enforced, but soon brought to a standstill by the commercial influences. It was not until three separate and distinct outbreaks had occurred that the sanitary authorities were given a free hand. It was not long after this that the last case of human plague occurred, and the city has since been free of plague.

Some features of the campaign against the rat are not without interest. During the time of the cases there were, on an average, 5,000 rats delivered daily to the various police precincts. The number in 1901 was about 2,000 per day. From Nov. 20, 1899, to Aug. 23, 1901, there had been examined 565,589 rats, and of these only 158 were infected with plague. The last plague rat was found on June 21, 1901, one year and five months after the last case in man. There were 69,718 rats examined in Kobe and Hiogo between May 1 and Aug. 23, 1901, and two infected rats found—the first was discovered in March, the last on June 11, 1901. The last plague case was Dec. 21, 1901.

The long time elapsing between the last plague case in man and in the rat is quite suggestive and seems to offer an explanation of the recrudescence of plague from time to time in which the source of infection is unac-

countable. Further, it emphasizes the fact that our measures against rat infection should be continuous, and for an almost indefinite time. In September, 1901, in conjunction with Dr. Bell of the Civil Hospital at Hong Kong, I made a bacteriologic examination of 251 dead rats which were collected by the scavenger force from all parts of that city. Without reference to locality where found, thirteen of these were dead from plague. Dr. Bell continued the examination of 100 each in October, November and December. He found for October 2 per cent., November 1 per cent., and for December none of the rats was infected. He says: "It is interesting to note that the subsidence of plague against human beings corresponds to that among rats. These examinations, while few, seem to establish the fact that one of the means, and what is believed to be the chief agency to not only disseminating plague but keeping it alive, is the rat. Certainly there was an intimate connection shown to exist between the rat infection and the cases of plague occurring among the better class of whites, who were living a considerable distance from the Chinese part of the city.

Whether the size or age of the rat has anything to do with the dissemination of plague, or whether it influences its virulence, can not be said. Of the number examined by us 124 were full grown, and of these 10 were infected; 63 were half-grown, and of these 3 infected. No young rats and mice were found infected.

We have already mentioned the fact that the temperature may have a direct influence in arresting plague, for it is a well-established fact that whenever the temperature reaches 81 to 83 F., and continues for some weeks, the plague mortality steadily diminishes. I have suggested that possibly the rat may be influenced in some way by the high temperature, maybe in a resting stage, and without inclination to migrate as at other seasons, and thereby may be less exposed to the infection and less liable to disseminate it.

Manila has now experienced two epidemics of plague since the American occupation. This focus of infection is almost as much import to the States as the Chinese and Japanese ports. In October, 1901, soon after my arrival, the sanitary authorities began a systematic campaign against plague, and particularly with reference to the destruction of vermin. All rats caught, killed or found dead were examined. The anti-rat campaign developed the same results there as it had done in Kobe and Osaka. Plague cases became fewer, even at the season when it had heretofore been the greatest number of cases. Unfortunately, just when it seemed that a full control had been attained, cholera made its appearance, and the anti-plague measures practically ceased. Plague then began to increase.

There is yet a diversity of opinion concerning the rôle played by suctorial insects in the transmission of plague from rat to rat, and from rat to man. Simond's contention appears to be established by Thompson in the epidemic of Sydney, but so far this is the only instance where observations of this character have been confirmatory. Laboratory experiments made in this country and in Europe appear to establish the fact that fleas from an infected rat are capable of transferring the pest bacillus to healthy rats. Terni claims that the *Pulex fasciatus* infests both rat and cat, which sometimes infests man, while Gautheir and Raybaud's experiments show that plague can be transmitted from rat to rat by the *Pulex fasciatus,* and by a smaller species resembling the *Pulex irritans,* also that the ascarides sometimes infesting rats can also transfer the bacillus from sick to healthy rats.

In eight experiments made with fleas which infest man, by placing these on rats they were able to transmit plague from rat to rat, all of them were successful. Tidswell's observations[1] rather confirm Simond's contention. He says: "It does not follow that fleas har-

1. This observation appeared after presentation of paper, Jour. Trop. Med., July 15, 1903.

bored by the rats in two widely different places would be exclusively the same species. With a view of determining this, a collection of fleas was made from rats coming under observation. Of 100 fleas 10 were *Pulex fasciatus,* 8 were *Typhlopsylla musculi,* 1 was *Pulex serraticeps,* and 81 were *Pulex pallidus. Pulex pallidus* has not been heretofore mentioned. This species, as well as *Pulex fasciatus,* bit men; the others did not." In view of the fact that our observations are not as yet either sufficiently numerous or accurate, it would be as well to include the destruction of all insect life in our measures against plague until it is conclusively proven that these have no bearing on its dissemination.

Now with regard to the prophylactic injections for plague, it has, as is well known, been extensively employed in India and has passed the experimental stages. The epidemic of plague in Kobe and Osaka demonstrated that it was of greatest value in preventing its spread. Statistics are tiresome, but are, notwithstanding, sometimes necessary. The number inoculated from Dec. 12, 1899, to Aug. 22, 1901, was 110,115 in Osaka, and 30,000 in Kobe.

While the prophylactic injections were not made obligatory, many persons availed themselves of them, especially those who had been in contact with plague cases or where plague rats were found on or near their domiciles, and those whose avocations would be more likely to be exposed to the infection. The police and rat collectors were all inoculated—several hundred—and none of these contracted plague. One physician died of pneumonic plague—he was not inoculated. This is quite in contrast to the rat coolies of Hong Kong, where many died of plague. The Yersin anti-pest serum was administered to all those who were in direct contact with cases, and in not a few instances to those who had come in contact with pest rats. In none of these did plague occur. From last accounts from Japan, the campaign of the sanitary surveillance still goes on, and it is the

intention to maintain this so long as there is danger of plague being a menace to that country.

The measures to be enforced in a port infected with plague must of necessity be modified in their details, according to the exigencies of the case. No better plan has been devised than has been adopted by the Japanese and Australian authorities in the management of their plague epidemics, which may be summarized as follows:

1. Detection of cases and of rats infected.
2. Provisions for the care and treatment of those sick.
3. Isolation and surveillance of contacts and suspects.
4. A general inspection, personal and domiciliary, of the place by a sufficient number of trained experts.
5. A general destruction and examination of vermin for plague.
6. The offer of prophylactic inoculations and immunization to all persons liable to be, or have been, exposed to plague.
7. Disinfection of personal effects and of domiciles presumably infected or liable to become infected by vermin.
8. Preventing persons exposed to plague leaving an infected place until free from infection.
9. A sanitary surveillance over the common carriers.
10. A coöperation between the sanitary authorities, national, state and municipal.
11. Publicity.

"Plague is regarded by nearly every citizen as a possibility elsewhere, but not in his own particular city. The same note of resentment is struck in nearly every place where it has appeared. In Oporto, Bombay, Alexandria, Osaka and San Francisco the same old story is repeated. The commercial interests resent the imputation, and those who have announced the unwelcome news have been regarded as public defamers of the city in which they live."[2]

There are a few notable examples now on record which stand out in vivid contrast to the above, as is instanced

2. British Med. Jour. Editorial.

by the action of the sanitary authorities of Japan, Sydney, Hong Kong, Glasgow and Mexico. Not only the simple announcement was made to the world, the regrettable fact of the appearance of plague in their city or country, but also that they were doing all that could be done to eradicate it. Whether success speedily crown their efforts or not did not rob them of the commendation, the assurance of coöperation or of the confidence and esteem of all countries.

It was indeed a lamentable fact that some of our citizens could not have seen their way clear to have been honest and upright, and to have adopted a proper course of action on dealing with plague soon after its discovery in San Francisco, instead of inviting disaster not only to themselves, but to the whole country, in their short-sighted policy of denial and suppression of the truth. We are confident that if the same amount of energy had been directed toward the eradication of plague as was displayed in attempts to obstruct and hamper the sanitary authorities, and in means of suppressing information, plague in the United States would have long since ceased to be a menace, and also there would not have been entailed the crime of sacrificing human lives for no other reason than to conserve and promote their commercial and political prestige.

It was a most humiliating spectacle! Misrepresentation, duplicity, fraud and suppression of truth were the watchwords emblazoned on their banners. The sanitary authorities of nation, state and city were opposed in everything they attempted to do. Attempts were made to suppress information, even reports sanctioned and made mandatory by the highest laws of our land, and in more than one instance their campaign was successful. There arose at one time what appeared to be the turning point in this tide of civic corruption, when reason and common sense would replace the foolish ostrich-like policy of the past, whereby the sanitary authorities would be allowed to discharge their duties

without further hindrance. But the representations made by these obstructionists were so plausible (?), their promises and agreements really having the smack of sincerity, that a few were so blinded by their glamour that they fell an easy prey, and, like Samson of old, blinded and manacled were led forth captive to furnish sport to be mocked and jibed by these modern Philistines. The simile here ends. The house still stood. But plague, like Banquo's ghost, would not down. It held the even tenor of its way, gradually extending its territory, despite the plan of campaign, and could not be exorcised either by executive bulls or judicial opinions, even when loudly proclaimed by a venial press.

It is believed that from the very first the now Public Health and Marine-Hospital Service had no other desire than to do all that was possible to eradicate plague from California, and, while mistakes have been made in directing its policy, no one will doubt its sincerity in sanitary work. But this service, like those of the state and city, was also incapable of escaping the clutches of the great organized combination of commercial and political interests; consequently its efforts came to naught. Probably more would have been accomplished in the beginning had it not undertaken too much and had been more mindful of the statutory and constitutional limitations between state and nation.

The same story was repeated over and over again, and no progress whatsoever was made in the eradication of plague. It was, so far as the combination was concerned, an endless chain of falsification, duplicity and sordid self-interest—a travesty on civic decency—a monumental disgrace. Just how long this condition of affairs was to continue, despite the protests of honest people, none could foresee. It appeared to resolve itself in awaiting a time when those of San Francisco and California could by some yet undiscovered means or new procedures be brought up out of the mire, and be made to see the error of their ways, and be compelled to do

something to eradicate the plague spot from their midst before it still further menaced the whole country. It did appear questionable whether anything would be permitted, until a sufficient number of cases had occurred to cause a panic and commercial disaster. Perhaps then they could be awakened from their self-satisfying dreams, would arise, clean their city, make the Chinese put their houses in order and keep them clean, or go.

The sanitary authorities of San Francisco and some of the state deserve the lasting gratitude of the nation. Too much praise can not be given to those who have constantly and fearlessly discharged their duties, despite the almost insuperable obstacles thrown in their way. The recently enacted law reorganizing the Marine-Hospital Service into a Public Health Service contained, among other things, a provision for a conference between its chief and the state health officials, to be held on the demand of five or more states or territories. Some of the health authorities, together with the writer, believed that steps should be immediately taken looking to a concerted action of the state and national authorities whereby plague could be eradicated.

The preliminary steps to this end were taken by the Conference of State and Provincial Boards of Heath at its annual meeting at New Haven in October, 1902. The resolutions unanimously passed by that body were of such character that a conference between the surgeon-general of the Public Health Service and the several states was a natural sequence. This conference was held in Washington early in January, 1903, and resolutions passed by this body reiterated those passed at New Haven, and further spoke in terms of no mistaken meaning to those who had persistently balked every well-meant effort to clean San Francisco's Chinatown of plague, that the time had come when they must cease "to quit their meanness," tell the truth and be good.

All these things they are doing or have promised to do—the very things which were suggested to them in

1900. Now the city board of health is not being interfered with. The state authorities, together with the United States Public Health and Marine-Hospital Service, are aiding it, and a hearty co-operation established between all, each doing its allotted part.

It does seem as if this is the beginning of the end, and that after conscientious and persistent effort that the danger of a further extension of plague will be dissipated, and that soon the painful memories of the past two years be sunk into oblivion. Let us hope that the era of good feeling, plain dealing, honesty of purpose, and above all, good citizenship, may have come, and be established among those who have heretofore been guiding the destinies of a great state.

DISCUSSION

ON THE PAPERS OF DRS. CALVERT AND KINYOUN.

Dr. F. G. Novy, Ann Arbor, Mich.—The history of plague, to my mind, forms one of the most interesting chapters in the history of epidemics. Nine years ago the telegraph reported the existence of plague in China, and when the news reached Europe there were very few physicians who had the slightest idea as to what the plague was. At all events, it was extending, passing through and beyond the chief empires of the Orient; in time it reached South America, North America, Africa, etc. At the outset France and Japan sent commissions to investigate the disease. These commissions were headed by Yersin and by Kitasato, respectively, and they worked independently and at the same time in Hong Kong. The announcement was soon made that they had both discovered the plague bacillus. As a matter of fact, to Yersin, and not to Kitasato, belongs the credit of this discovery.

In plague we have a well-defined disease, caused by a well-defined organism; this organism can be seen easily under the microscope and can be easily found in every case brought to the postmortem table; and, moreover, it can be cultivated without difficulty. Experiments can be made on animals and the relation of the germ to the disease can be demonstrated to anyone.

The question naturally comes up: "How does it get into the body?" There is one point which I should like to emphasize. and that is, that plague is essentially an animal disease and is primarily not a human disease. It affects by preference animals, rats or other rodents in China, and these animals introduce the disease into the human subject. How? The flea

is commonly held responsible for such transmission, which, however, is by no means absolutely proven. To my mind, other insects may take a part in and other means exist for its conveyance. The usual mode of entry is presumably through an inoculation wound, but it is exceedingly difficult to indicate just where the bacillus enters the body.

As to the germ, we may say that its virulence decreases when it is kept in ordinary culture. The organism is not so very resistant and is readily destroyed by various injurious conditions and substances. Several types of the disease are met with according to the manner of entrance of the germ. The most common type, where the lymphatic glands are enlarged, the so-called bubonic form of plague, probably results as I have indicated, by inoculation through wounds. The pneumonic type follows the entrance of the germ along the respiratory passages. A third or septicemic variety is at times met with. The mode of entrance, the virulence of the germ and the resistance of the individual are determining factors in the production of a given type of the disease.

I wish, though, to especially emphasize the prevention of plague by vaccination methods. In addition to these means we have, of course, methods of disinfection, the isolation of the sick, the segregation of those exposed; all of which are powerful means for controlling the disease. There are two methods of vaccinating against this disease: First is Haffkine's vaccine, which is prepared by growing the plague germ in beef tea. When such cultures are fully developed the germs are killed by exposure to heat, and the material thus obtained is ready for injection into the human subject. The immunity thus acquired by the individual is fairly lasting. A second method of vaccination consists in the use of Yersin's serum. This, when injected into the human subject, has a curative as well as an immunizing action. It is prepared by injecting plague bacilli, of the most virulent type that can be obtained, into the horse. The animal thus immunized gives a serum which is strongly protective, but the duration of such immunity is not as long as when Haffkine's vaccine is used. I believe that Yersin's serum, when properly prepared and not too old, when used in sufficient amount and in the right way, will give perfect results. Two years ago we had a case of plague infection of accidental origin in the laboratory. One of the students, in some unknown way, inoculated himself while engaged in making vaccines. He developed the pneumonic type of plague. The onset of the disease was sudden and severe. My attention was called to his illness about twenty hours after he had taken to bed. Pronounced evidences of the disease were found, such as high temperature, anxiety, respiratory distress, etc. An injection of the Yersin serum was given at once. Preparations were made at once

from the throat and from the material coughed up, but these showed no germs. Within the next two or three hours a blood-streaked particle of sputum gave positive evidence of the existence of plague. The fellow was removed from his companions and taken to an isolation hospital. His condition was unfavorable to say the least. The young man had the pneumonic type of the disease, which form has a mortality of just about 100 per cent. One hundred and twenty c.c. of Yersin's serum were injected into the patient; of this 60 c.c. were given intravenously and 60 c.c. subcutaneously. Within twenty-four hours the temperature dropped from 104.5 to 100 and he improved greatly. Apart from a marked cardiac weakness which persisted for several months, he made an uneventful recovery. This case clearly shows the efficiency of the serum when given in large quantities and right into the circulation.

In connection with this subject I should like to say that it is exceedingly difficult to find the first case of plague which appears in any locality, simply because we are not expecting it and because it may be mistaken for several other diseases. It is very essential that the utmost care should be exercised in looking over suspicious cases, especially when one is in a locality where the disease is not known to exist. Early recognition means a relatively easy eradication. When, however, plague does establish itself it is exceedingly difficult to stamp out. We know that there is hardly another disease which is so difficult to eradicate as this one, and this is probably due to the fact that the germ lives in the animal body and thus keeps up a local focus for the spread of the disease.

Dr. Emmet Rixford, San Francisco—I do not know why I have been called on to speak before this meeting as I do not pretend to have a knowledge of bubonic plague. Though I reside in San Francisco I have never seen a case. If, however, you call on me as a sort of representative of San Francisco to say something from her side on this matter of the alleged concealment of the presence of the plague, a matter which has filled the medical and lay press for many months, I might be emboldened to occupy your attention for a few moments. San Francisco is not proud of the position in which she finds herself obliged to bear the taunts and sharp criticism of the guardians of the public health on the Atlantic coast. But there is something to be said on her side. She feels that she has not been quite fairly treated. It is, of course, hard for you to appreciate that much of the trouble was due to the local political situation in which you can have no interest, but such is the fact. There is no doubt in the minds of most of our local profession that had it not been for the extreme positions taken by rival and opposing political factions the sensational elements of the matter would have been largely avoided. The statements which received greatest publicity

were exaggerated and far from the truth on both sides; the great mass of substantial citizens and the conservative portions of the medical profession were not heard in the controversy. Much has been said as to the domination of the commercial interests. Certainly the Merchants' Association of San Francisco, which is representative, did not at any time deny the existence of the plague. What San Francisco feared was quarantine. What she denied was the justification of quarantine, which was not only threatened, but in the case of two states was actually inaugurated against the state of California. At no time have the facts in the case warranted even the threat of quarantine. Quarantine is a frightful punishment to mete out against a community; it is justified only in the demonstrated presence of widespread and progressing epidemic. I am told to-day that the boards of health which met not long since had no real intention of placing quarantine on the state of California, that they used the threat merely to bring us to our senses and that the matter of possible quarantine would never have been suggested had the medical profession of California openly acknowledged the presence of the plague in San Francisco instead of denying it. The facts are that the medical profession of California in session of its State Medical Society two years ago did, by resolution, put itself on record as recognizing the presence of the plague in San Francisco, and the fact was widely published in the medical and lay press. It is now three years since the plague was discovered in San Francisco, and in all that time there have been but 90 sporadic cases, and these, with but two or three exceptions, confined to the Chinese race. At no time has there been an epidemic of plague in San Francisco, or anything approaching it.

There is another thing which seems to me to have considerable meaning in extenuation of this unfortunate controversy, and that is the fact that the world knows a great deal more about the plague to-day than it did even two or three years ago, and that had the present knowledge of the subject been generally disseminated when the plague first appeared in San Francisco the efficiency of preventive measures would have been appreciated, the comparatively slight contagious characters of the disease would have been recognized and the hysterical statements which set the whole United States agog would not have been made, and intelligent effort would earlier have been directed to the eradication of the disease. Happily now the situation in San Francisco is more satisfactory; there has not appeared a single case for more than two months, and the officials of the United States Marine-Hospital Service and the local and state boards of health are working in harmony, watching and fighting the disease, and it is to

be hoped that we can soon say that the disease has no longer a focus in San Francisco.

Dr. W. J. Calvert, St. Louis—There is one feature of prophylaxis of plague which I should like to mention, namely, the dangers of Oriental infected centers or plague centers. In Canton and Hong Kong plague exists from year to year with practically no steps taken to check its propagation and dissemination. The present pandemic practically originated in Canton, to be distributed from Hong Kong to other parts. Even if plague could be quickly and effectually eradicated from San Francisco, Mexico, etc., it would soon be reintroduced from Hong Kong. It seems to me that in discussions on the prophylaxis of plague too little attention has been given to these sources of the disease. As long as they exist the world is exposed to infection. I think we should try to improve the hygienic conditions in these centers by properly educating the Chinese, who at present know nothing of modern medicine. If properly approached they would be only too willing to lend a helping hand to rid their people of this pest, and at the same time the outside world from the danger of infection.

EXECUTIVE SESSION

TUESDAY, MAY 5.

Address of the Chairman, W. S. Thayer, Baltimore.

"Restorative Influence of Ozone on the Blood." G. Lenox Curtis. Discussed by F. A. Weber, Milwaukee.

"The Boy's Venereal Peril." Ferd. C. Valentine, New York. Discussion by J. J Walsh, New York; F. W. Robbin, Michigan; DeLancey Rochester, Buffalo, N. Y.; Theodore Potter, Indianapolis; M. M. Smith, Texas; Frank A. Jones, Memphis; E. L. Stevens, Iowa; W. Shropshire, Texas, and Ferd. C. Valentine.

"Continued Fever, Neither Malarial nor Typhoid; a Clinical Study." T. J. Happel, Trenton, Tenn.

"Southern Fevers." William Krauss, Memphis, Tenn.

"Clinical Observation of Ninety Cases of Typhoid Fever, with Four Deaths, with Special Reference to Therapeutic Fasting." R. M. Harbin, Rome, Ga.

"Typhoid Fever at High Altitudes, with a Résumé of 600 Cases." J. N. Hall, Denver.

Discussion on the above four papers by T. B. Futcher, Baltimore; James Tyson, Philadelphia; J. A. Witherspoon, Nashville, Tenn.; C. F. Mason, U. S. A.; R. W. Lindson, Little Rock, Ark. Discussion of last four papers to be continued with the Symposium on Typhoid Fever on Thursday morning.

The Committee on Nominations was announced as J. H. Musser, Philadelphia; William Krauss, Memphis, and Thomas McCrae, Baltimore.

WEDNESDAY, MAY 6.

"Weakness and Dilatation of the Heart Due to Chronic Nutritional Diseases." G. W. McCaskey, Fort Wayne, Ind. No discussion.

"Limitations of Non-Surgical Treatment in Intestinal Obstruction." J. R. Eastman, Indianapolis. No discussion.

"The Occurrence of Strongyloides Intestinalis in the United States." M. L. Price, Baltimore. Discussion by C. W. Stiles, Washington, D. C.; A. J. Smith, Galveston, Texas; A. P. Ohlmacher, Ohio, and J. T. Moore, Galveston, Texas.

"Clinical Diagnosis of Intestinal Parasites." C. W. Stiles, Washington, D. C.

"Uncinariasis in the United States, with Especial Reference to Its Occurrence in Texas." Allen J. Smith, Galveston, Texas.

"Treatment of Uncinariasis." T. A. Claytor, Washington, D. C.

Discussion on the last three papers by W. G. Harrison, Alabama; E. D. Bondurant, Mobile, Ala.; C. A. Mohr, Alabama; G. W. McCaskey, Fort Wayne, Ind.; T. A. Williams, Edinburgh, Scotland; Claude A. Smith, Atlanta, Ga.; James B. Herrick, Chicago; William Britt Burns, Memphis, Tenn.; C. W. Stiles, Washington, D. C.; A. J. Smith, Galveston, Texas, and T. A. Claytor, Washington, D. C.

"Bacillary Dysentery." C. F. Mason, Fort Sam Houston, Texas.

"Contribution to the Study of the Summer Diarrheas of Infancy." J. Mason Knox, Baltimore.

"Malarial Dysentery." William Britt Burns, Memphis, Tenn.

"The Clinical History and Pathology of Amebic Dysentery." H. F. Harris, Atlanta, Ga.

"A Study of the Cases of Amebic Dysentery Occurring at the Johns Hopkins Hospital." T. B. Futcher, Baltimore.

"The Treatment of Acute Dysentery." J. M. Anders, Philadelphia.

Discussion on the above six papers by Alexander Lambert, New York; T. B. Dabney, New Orleans; J. T. Moore, Galveston, Texas; W. Shropshire, Yoakum, Texas; C. F. Mason, Fort Sam Houston, Texas; J. H. Mason Knox, Baltimore; William Britt Burns, Memphis, Tenn.; H. F. Harris, Atlanta, Ga.; T. B. Futcher, Baltimore; J. M. Anders, Philadelphia.

"Abdominal Pain in Pleurisy and Pneumonia." James B. Herrick, Chicago. Discussion by J. N. Hall, Denver; T. McCrae, Baltimore; J. M. Anders, Philadelphia; B. W. Sippey, Chicago, and J. B. Herrick, Chicago.

"Traumatic Pneumonia." W. T. English, Pittsburg, Pa.

THURSDAY, MAY 7.

COMBINED MEETINGS WITH SECTION ON HYGIENE AND SANITARY SCIENCE.

"The Sources and Manner of Infection of Typhoid Fever." William H. Welch, Baltimore.

"Prophylaxis of Typhoid Fever." John S. Fulton, Baltimore.

Discussion on the above two papers by John H. Musser, Philadelphia; DeLancey Rochester, Buffalo, N. Y.; William Krauss, Memphis; Frank Warner, Ohio; J. N. Hurty, Indianapolis; Sewell, Denver; E. J. Roberts, Nashville, Tenn.; C. F. Adams, New Jersey; James Tyson, Philadelphia; J. J. Walsh, New York; Walter Shropshire, Texas; T. J. Happel, Trenton, Tenn., and John S. Fulton, Baltimore.

"The Mode of Transmission of Yellow Fever." James Carroll, U. S. A. Discussion by Stanford E. Chaillé, Edmond

Souchon, Q. Kohnke and Felix Formento, New Orleans; C. W. Stiles, Washington, D. C., and James Carroll, U. S. A.

The following officers were elected: Chairman, Alexander Lambert, New York; secretary, J. S. Miller, Chicago.

"Sources and Manner of Infection of Plague." W. J. Calvert, St. Louis.

"Prophylaxis of Plague." J. J. Kinyoun, Philadelphia.

Discussion on the two above papers by F. G. Novy, Ann Arbor, Mich.; E. Rixford, San Francisco, and W. J. Calvert, St. Louis.

"Arthritis Deformans." Thomas McCrae, Baltimore. Discussion by J. J. Walsh, New York; J. T. Moore, Galveston, Texas; C. E. Skinner, New Haven, and Thomas McCrae, Baltimore.

"Albuminuria in Diabetes; Its Etiology and Importance as a Factor in the Production of Diabetic Coma." Arthur R. Elliott, Chicago. Discussed by William Krauss, Memphis, Tenn.; F. R. Weber, Milwaukee, and A. R. Elliott, Chicago.

FRIDAY, MAY 8.

A committee to appoint a member to the House of Delegates was named as follows: H. L. Sullivan, Mississippi; William Krauss, Tennessee, and T. B. Futcher, Maryland. They reported in favor of Frank A. Jones, Tennessee.

"The Passing of Chronic Rheumatism." J. J. Walsh, New York. Discussion by James Tyson, Philadelphia; Thomas McCrae, Baltimore; Alexander Lambert and J. J. Walsh, New York.

"Hemoglobinuric Fever; Its Cause and Treatment." Walter Shropshire, Yoakum, Texas. Discussion by William Krauss, Memphis, Tenn.; Barnes, Louisiana; Frank A. Jones, Memphis, Tenn.; Southerland, Mississippi; L. A. Jabra, Tennessee; James B. McElroy, Mississippi; W. S. Thayer, Baltimore; Williams, Edinburgh, Scotland, and Walter Shropshire, Texas.

"Some Clinical Aspects of Tubercular Peritonitis." Joseph Eichberg, Cincinnati. No discussion.

"Tuberculosis in the Negro." Seale Harris, Union Springs, Ala. No discussion.

"Should the Tuberculosis Patient Know the Truth Regarding His Condition?" C. P. Ambler, Ashville, N. C. No discussion.

Adjourned.

BOSTON MEDICAL LIBRARY
MAR 16 1928

INDEX

Abdominal pain in pleurisy and pneumonia, 259
Adams, C. F., discussion on typhoid fever, 501
Albuminuria in diabetes mellitus, 380
Ambler, C. P., the tuberculous patient, should he be informed as to his condition, 464
Anders, J. M., acute dysentery, 244
Anders, J. M., discussion on abdominal pain, 274
Anders, J. M., discussion on dysentery, 257
Arthritis deformans, 331
Bondurant, E. D., discussion on papers of Drs. Stiles, Smith and Claytor, 152
Boy's venereal peril, the, 20
Brown, E. J., discussion on tubercular peritonitis, 446
Burns, W. B., discussion on dysentery, 256
Burns, W. B., discussion on papers of Drs. Stiles, Smith and Claytor, 155
Burns, W. B., malarial dysentery, 197
Calvert, W. J., discussion on plague, 550
Calvert, W. J., sources of plague, 506
Carroll, J., discussion on transmission of yellow fever, 329
Carroll, J., transmission of yellow fever, 291
Chaillé, S. E., stegomyia and fomites in yellow fever, 308
Chairman's address, 9
Claytor, T. A., discussion on papers of Drs. Stiles, Smith and Claytor, 160
Claytor, T. A., treatment of uncinariasis, 142
Clinical medicine, observations on teaching of, 9
Colorado, typhoid fever in, 61
Continued fever neither malarial nor typhoid, 31
Cooper, C. E., typhoid fever in Colorado, 61
Crook, J. L., discussion on fevers, 73
Dabney, T. S., discussion on dysentery, 253
Diabetes mellitus, albuminuria in, 380
Discussion on abdominal pain in pleurisy and pneumonia, 273
Discussion on albuminuria in diabetes, 391
Discussion on arthritis deformans, 377
Discussion on chronic rheumatism, 403
Discussion on Dr. Valentine's paper, 26
Discussion on dysentery, 253
Discussion on fevers, 71
Discussion on hemoglobinuric fever, 423
Discussion on papers of Drs. Stiles, Smith and Claytor, 150
Discussion on plague, 546
Discussion on strongyloides intestinalis, 133
Discussion on transmission of yellow fever, 322
Discussion on tubercular peritonitis, 446
Discussion on typhoid fever, 494

Dysentery, acute, treatment of, 244
Dysentery, amebic, pathology and clinical history of, 201
Dysentery, amebic, study of cases of, occurring at Johns Hopkins Hospital, 213
Dysentery, bacillary (Shiga) 161
Dysentery, malarial, 197
Eastman, J. R., acute intestinal obstruction, 87
Eichberg, J., tubercular peritonitis, 429
Elliott, A. R., albuminuria in diabetes mellitus, 380
Elliott, A. R., discussion on albuminuria in diabetes, 392
English, W. T., traumatic pneumonia, 277
Executive session, 551
Formento, F., discussion on transmission of yellow fever, 327
Fulton, J. S., discussion on typhoid fever, 504
Fulton, J. S., typhoid fever, 470
Futcher, T. B., amebic dysentery, 213
Futcher, T. B., discussion on dysentery, 257
Futcher, T. B., discussion on fevers, 71
Hall, J. N., discussion on abdominal pain, 273
Hall, J. N., typhoid fever in Colorado, 61
Happel, T. J., continued fever, 31
Happel, T. J., discussion on typhoid fever, 503
Harbin, R. M., typhoid fever, 52
Harris, H. F., amebic dysentery, 201
Harris, H. F., discussion on dysentery, 256
Harris, S., tuberculosis in negro, 447
Harrison, W. G., discussion on papers of Drs. Stiles, Smith and Claytor, 150
Heart weakness and dilatation in chronic nutritional disease, 74
Hemoglobinuric fever, 407
Herrick, J. B., abdominal pain in pleurisy and pneumonia, 259
Herrick, J. B., discussion on abdominal pain, 275
Herrick, J. B., discussion on papers of Drs. Stiles, Smith and Claytor, 155
Hurty, J. N., discussion on typhoid fever, 498
Intestinal obstruction, acute, limitations of non-surgical treatment of, 87
Intestinal parasites, clinical diagnosis of, 137
Jones, F. A., discussion on Dr. Valentine's paper, 28
Jones, F. A., discussion on hemoglobinuric fever, 424
Kinyoun, J. J., prophylaxis of plague, 516
Knox, J. H. M., discussion on dysentery, 255
Knox, Jr., J. H. M., summer diarrheas of infancy, 176
Kohnke, Q., discussion on transmission of yellow fever, 326
Krauss, W., discussion on albuminuria in diabetes, 391
Krauss, W., discussion on hemoglobinuric fever, 423
Krauss, W., discussion on typhoid fever, 496
Krauss, W., southern fevers, 42
Lambert, A., discussion on chronic rheumatism, 404
Lambert, A., discussion on dysentery, 253
Lindsey, R. W., discussion on fevers, 73
McCaskey, G. W., heart weakness and dilatation, 74
McCaskey, G. W., discussion on papers of Drs. Stiles, Smith and Claytor, 153
McCrae, T., arthritis deformans, 331
McCrae, T., discussion on abdominal pain, 274

McCrae, T., discussion on arthritis deformans, 379
McCrae, T., discussion on chronic rheumatism, 404
McElroy, J. B., discussion on hemoglobinuric fever, 425
Mason, C. F., bacillary dysentery, 161
Mason, C. F., discussion on dysentery, 255
Mason, C. F., discussion on fevers, 73
Mohr, C. A., discussion on papers of Drs. Stiles, Smith and Claytor, 153
Moore, J. T., discussion on arthritis deformans, 378
Moore, J. T., discussion on strongyloides, 135
Moore, J. T., discussion on dysentery, 254
Musser, J. H., discussion on typhoid fever, 494
Negro, tuberculosis in, 447
Novy, F. G., discussion on plague, 546
Ohlmacher, A. P., discussion on strongyloides, 135
Plague, prophylaxis of, 516
Plague, sources of, 506
Peritonitis, tubercular, some clinical aspects of, 429
Pneumonia, traumatic, 277
Potter, T., discussion on Dr. Valentine's paper, 27
Price, M. L., strongyloides intestinalis in U. S., 100
Rheumatism, chronic, passing of, 393
Rixford, E., discussion on plague, 548
Robbins, F. W., discussion on Dr. Valentine's paper on boy's venereal peril, 26
Roberts, D. J., discussion on typhoid fever, 500
Rochester, D., discussion on Dr. Valentine's paper, 27
Rochester, D., discussion on typhoid fever, 496
Sewall, H., discussion on typhoid fever, 499
Shropshire, W., discussion on dysentery, 254
Shropshire, W., discussion on Dr. Valentine's paper, 29
Shropshire, W., discussion on hemoglobinuric fever, 427
Shropshire, W., discussion on typhoid fever, 502
Shropshire, W., hemoglobinuric fever, 407
Skinner, C. E., discussion on arthritis deformans, 378
Smith, A. J., discussion on papers of Drs. Stiles, Smith and Claytor, 157
Smith, A. J., discussion on strongyloides, 134
Smith, C. A., discussion on papers of Drs. Stiles, Smith and Claytor, 154
Smith, M. M., discussion on Dr. Valentine's paper, 28
Souchon, E., discussion on transmission of yellow fever, 322
Southerland, H. L., discussion on hemoglobinuric fever, 424
Southern fevers, 42
Stegomyia and fomites in yellow fever, 308
Stephens, E. L., discussion on boy's venereal peril, 29
Stiles, C. W., discussion on papers of Drs. Stiles, Smith and Claytor, 156
Stiles, C. W., discussion on strongyloides, 133
Stiles, C. W., discussion on transmission of yellow fever, 328
Stiles, C. W., intestinal parasites, 137
"Still's disease," 365
Strongyloides intestinalis, occurrence of, in United States, 100
Summer diarrheas of infancy, contribution to study of, 176
Thayer, W. S., Chairman's address, 9
Thayer, W. S., discussion on hemoglobinuric fever, 426

Tuberculosis in the negro, 447
Tuberculous patient, should he know the truth regarding his condition, 464
Typhoid fever, 470
Typhoid fever, clinical observation of 90 cases of, 52
Typhoid fever in Colorado, 61
Tyson, J., discussion on chronic rheumatism, 403
Tyson, J., discussion on fevers, 71
Tyson, J., discussion on tubercular peritonitis, 446
Tyson, J., discussion on typhoid fever, 501
Uncinariasis, treatment of, 142
Valentine, F. C., boy's venereal peril, 20
Valentine, F. C., discussion on boy's venereal peril, 29
Walsh, J. J., discussion on arthritis deformans, 377
Walsh, J. J., discussion on chronic rheumatism, 405
Walsh, J. J., discussion on Dr. Valentine's paper on boy's venereal peril, 26
Walsh, J. J., discussion on typhoid fever, 501
Walsh, J. J., passing of chronic rheumatism, 393
Warner, S., discussion on typhoid fever, 497
Weber, F. R., discussion on albuminuria in diabetes, 391
Witherspoon, J. A., discussion on fevers, 72
Yellow fever, stegomyia and fomites in, 308
Yellow fever, transmission of, 291

41
6237